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CEREBRAL PALSY: CAUSES AND EARLY DIAGNOSIS*

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At one time it was considered that birth injury was the commonest cause of cerebral palsy. Today, antenatal factors are accepted as the main cause. The antenatal factors concerned are either genetic, such as primary mal-development of the embryonic brain, or illness in the mother.

The stimulating study made in Australia[1] of the effect of German measles on the heart, eyes and ears of the embryo in the first 3 months of gestation has resulted in the discovery that other illness in the mother may be potent causes of congenital malformations. The brain is the most vulnerable of all body tissues. Any condition in the mother that interferes with the supply of oxygen to the foetus, such as antenatal haemorrhage or toxaemia of pregnancy, may damage the developing brain. In addition, illnesses in the mother, such as German measles, other virus diseases or even ordinary ailments, may have a direct or indirect effect on the developing organism.

However, in spite of all this extra knowledge that has been gained in the last decade, it is still uncertain when any disturbances such as those mentioned above are responsible for a developmental abnormality. Swan[2] estimates that the risk ranges from 83% when infection occurs in the 1st month to 61% in the 4th. In the last 5 months of gestation the risk declines from 29 to 11%.

It is not everyone who accepts these figures and many cast doubts on this high incidence. Amongst the 131 cerebral palsy cases of my series, 4 (3%) had had German measles. Doctors who have experience in obstetrics will be able to recall any number of cases of antenatal bleeding, and toxaemia of pregnancy, that resulted in perfectly normal children. Hence it is most difficult to be exact in one's inferences. However, one cannot ignore the statistical possibilities and it is quite certain that, in a fairly high proportion of patients suffering from cerebral palsy, the cause must be looked for during the period of gestation and not at birth.

*A paper presented at the South African Medical Congress, Pretoria, 1955.

An analysis of the factors involved in 131 of my own cases of cerebral palsy in which details were obtained is shown below:

Prematures (under 5½ lbs.)	37	(28%)
Prematures (over 5½ lbs.)	5	(4%)
Post-mature	5	(4%)
ANTENATAL:		
A. *Uncomplicated gestation*	66	(50%)
B. *Complicated gestation:*		
(One case may show more than one complication)		
Antenatal haemorrhage	14	(11%)
Toxaemia of pregnancy	12	(9%)
Rh factor (with kernicterus)	10	(7%)
German measles	4	(3%)
Vomiting (excessive for 9 months)	4	(3%)
Malaria	2	(1·5%)
Pyelitis	2	(1·5%)
PERINATAL:		
A. *Normal birth*	79	(60%)
B. *Abnormal birth:*		
Forceps delivery	10	(7%)
Rapid delivery	10	(7%)
Prolonged delivery	7	(5%)
Abnormal presentation (breech 3, face 1, P.O.P. 1)	5	(4%)
Caesarean section	4	(3%)
C. *Abnormalities at birth:*		
Cyanosis	18	(14%)
Asphyxia	5	(3%)
Cord tight round neck	4	(3%)
Severe illness	4	(3%)
Haemorrhage	1	(·75%)
POSTNATAL:		
Encephalitis	11	(8%)
Meningitis	3	(2%)
Head injuries	1	(·75%)
Embolus	1	(·75%)
Gross cerebral abnormalities:		
Microcephalic	1	
Encephalocele	1	
Mongol	1	

The above figures are not a true reflection of the proportion of factors involved in the consideration of cerebral palsy. In quite a number of instances more than one defect was present. For instance, a premature baby may well have had an abnormal birth, being as a result cyanosed and asphyxiated. Its mother may have suffered from toxaemia or antenatal haemorrhage. The prematurity itself may have been a potent contributory factor to the cerebral palsy. At birth there may have been haemorrhage or direct injury to the brain. How to assess these factors is extremely difficult.

Winthrop M. Phelps[3] made a very interesting contribution on 'mid-line defects' in the brain, showing how congenital defects in the mid-line of the brain may cause cerebral palsy of a spastic or athetoid type, depending on whether it involved the cortex or the basal nuclei. The first 3 months of pregnancy must be studied in great detail to find the cause in some of these cases. These are the types of cases that may give rise to difficulties at birth and the ultimate disability is then attributed to birth injury.

It is to be noted that haemorrhage in the brain at birth is unlikely to produce an absolutely symmetrical lesion because the area of haemorrhage is unlikely to be absolutely equal on both sides of the brain. However, anoxaemia may be the cause of a symmetrical bilateral lesion.

I do not think that with the present state of our knowledge it is possible to make an accurate assessment of the numbers developing cerebral palsy as a result of birth injury or those due to antenatal or genetic factors.

We can be quite definite about the postnatal factors, about those born with gross deformities of the skull, about the cases of kernicterus. We are justified in assuming that cerebral palsy in a child whose mother has had a perfectly normal pregnancy and labour is probably due to a developmental abnormality. In my series, out of 56 perfectly normal pregnancies which preceded the birth of children who developed cerebral palsy, 36 (64%) ended in perfectly normal confinements. This confirms the fact that the majority of cases were not due to birth injury. Pohl[4] states that in his clinic only 5% are due to birth injury and Dr. M. Medalie[5] states that at the Forest Town School for Spastics in Johannesburg only about 4% were due to this factor. In my series a much larger incidence occurs.

One important and essentially modern factor that in the past has not operated are children saved from tubercular meningitis. I have had several cases left with various degrees of spasticity. There must surely be quite a number of such cases in the country, now that the recovery rate is so high.

DIAGNOSIS OF CEREBRAL PALSY

The vast majority of fully developed cases of cerebral palsy are easy to diagnose—the spastics are diagnosed by the signs of an upper motor neurone lesion, the athetoids by the purposeless involuntary movements of their limbs and face, the ataxics by the nystagmus, scanning speech, lack of balance and flaccidity.

The difficult cases to diagnose are those in early infancy, when it is so important to make a diagnosis—the cases with minimal signs and those with mental retardation who show no classical signs. The differentiation between athetosis with tension and spasticity may be extremely confusing. Evans[6] in 1946 reported that of 50 consecutive cases admitted to Carshalton, 54% were athetoid and 40% spastic, while the pre-admission diagnosis gave spasticity in 92% cases.

Age of Infant

It is commonly accepted that it is impossible to diagnose cerebral palsy before 2 years of age. That is not true. It should be possible to diagnose it long before that and prevent a large number of the deformities that occur. Two very young infants have been seen recently which will well illustrate the problem. The details of the former only will be given as the signs and symptoms were very similar in both.

Case Report. A female child aged 11 weeks was brought from the country as a feeding problem. The child had been vomiting and crying since birth. Breast milk was vomited just as readily as 8 different types of artificial food that were subsequently tried. The mother thought that the child might be unable to see. She also stated that the child was holding herself rather stiffly and that she had great difficulty in dressing her. No definite opinion was given at that time, for it was extremely difficult to examine a child that was crying and holding herself stiff. Advice on feeding was given and a sedative ordered.

The child was brought back 4 weeks later with no improvement. On this occasion detailed examinations of her eyes were made by several ophthalmologists. It was felt that the child could see, but the amount of vision present was uncertain. The light reflex was present.

It was then noticed that her limbs were unusually stiff and that it was very difficult to put a napkin on. There was marked adductor spasm and a stretch reflex was present at the knees. Close observation for a few days revealed the true state of affairs.

It had by this time become obvious that she was a case of cerebral palsy—either a rigid type or one developing spasticity—and that all her signs and symptoms could be explained on that basis.

Out of 65 of my series of cases, 40 (62%) had their disability noticed by their parents when under 1 year of age. Unfortunately only 3% were diagnosed in the 1st year, and 10% by the end of the 2nd year. It is obvious that medical students must be made more conscious of the existence of cerebral palsy. I may say that the presence of the three cerebral palsy schools in South Africa has been responsible for the dissemination of a considerable amount of information to students and doctors on this subject.

There is initially no abnormality of the muscle fibres—the muscles can function normally if normal stimuli are sent to them. To diagnose cerebral palsy in the early stages it is necessary to compare the reactions of a normal baby with those of a baby with cerebral palsy.

A normal baby's movements in the first few months of life are involuntary. In due course, their place is taken by voluntary movements. The normal baby's brain constantly receives external and internal stimuli which help it to build up a mental picture of motor action and hence initiate movements to effect the particular action shown in the picture. The movement produced supplies further stimuli. A baby who does

not receive these stimuli, owing to the cutting off of the motor paths, cannot make a complete mental picture and consequently cannot attempt the complete appropriate movements. When the baby does receive stimuli he tries to make the movements by damaged motor paths—so the movements are abnormal. Once one can get the baby to build up a mental picture of the movement required and can show him what the movements should be, he will compensate with his motor paths. This will enable him to perform the motor acts more normally, though still imperfectly.

We owe a great deal to Dr. Arnold Gesell of Yale, whose studies of the normal infant have enabled us to appreciate the abnormal.

Diagnostic Features of Spastic Types

The musculature involved in this type may be generalized or localized. The following signs are to be looked for:

Tongue may have restricted movements. Sucking difficulties are frequent early signs.

Swallowing is frequently difficult and dribbling is a prominent feature.

Eye movements are restricted and an object may not be followed to the periphery at a time when this would be done by a normal child. A squint is not infrequent.

Hearing defects are not common.

Smiling is often delayed. Normally it is present at 4-6 weeks.

The control of the *head and neck* muscles is frequently delayed beyond the 3rd month, and that of the arms and legs beyond the 4th month.

A normal baby usually sits at 6 months, whereas a cerebral palsied child may not sit alone even after the age of 1 year. The same applies to the other milestones of development—crawling, standing, walking and talking.

Aversion of the head whilst prone is quite a common characteristic of a normal infant. The cerebral palsied child lies helpless, cannot crane his neck, or if prone avert his head. Such a child may be suffocated because it cannot move.

The grasp and the Moro reflexes, which normally disappear by the 4th month, may be retained for a considerable time.

The foetal position of the back (one curve) can be noticed for several years.

A stretch reflex at the elbow and knees may be elicited and an adductor spasm of the thighs may be present.

Some of the children's abnormalities are best shown by suspension in different positions.

Lifting by armpits. A cerebral palsied baby will hold its legs straight or scissored with toes pointing down, whereas a normal child will flex knees and hips (Fig. 1).

Holding upside down by legs. In a normal child the arms drop and the head extends reflexly, whereas in a cerebral palsied child the arms do not drop (or only one drops) and the head does not extend (Fig. 2).

Lower limbs. If one leg is held by the ankle, the other will remain extended, whereas in a normal child the free leg will be flexed (Fig. 3). When the free leg is forced into flexion it will remain flexed in the normal child, but in spastic or rigid limbs it will usually revert to the extended position (Fig. 4). A detailed and interesting description of these signs are given by Eirenne Collis.[7]

Diagnostic Features of Athetoid Type

This is a more difficult condition to diagnose, and can only be recognized at a later stage than the spastic type.

Fig. 1. Suspension tests in cerebral palsy. Lifting by armpits. On right, normal child: limbs all flexed and freely moveable. On left, child with spastic quadriplegia: lower limbs in adductor spasm with plantar flexion of feet; tendency to 'scissoring'.

Fig. 2. Suspension tests in cerebral palsy. Suspension by feet. On right, normal child: upper limbs reach down to ground. On left, child with spastic quadriplegia: upper limbs stiff and held at chest level.

Fig. 3. Suspension tests in cerebral palsy. Holding by one foot. On right, normal child: free leg flexed. On left, child with spastic quadriplegia: free leg extended and stiff.

Fig. 4. Suspension tests in cerebral palsy. Children held by right foot, the free left leg being pushed down. On right, normal child: it is easy to flex whole limb and it remains flexed. On left, child with spastic quadriplegia: limb flexed with difficulty and soon resumes its extended stiff position.

Athetoid movements are best noticed when the child tries to do anything, but frequently they can be seen even at rest.

The tongue shows irregular movements with resulting difficulties in swallowing, sucking or speaking. Dribbling is present. There is no adductor spasm or stretch reflex, and the head is not extended.

The milestones of development are delayed.

Hearing defects are relatively frequent. In athetosis due to the Rh factor this is quite common.

Diagnostic Features of Ataxic type

These are not usually noticed until the child has to balance himself. There may be marked muscular flaccidity. Nystagmus is usually present, but not necessarily.

Diagnostic Features of Rigid Type

The rigid type may resemble the spastics and have many of their disabilities. The reflexes however, are, usually normal and the stiffness may be variable. This condition may be commoner than is usually thought. Eirenne Collis[7] describes it very interestingly. She states that it is always associated with mental defectiveness.

Differential Diagnosis:

1. Simple mental retardation.
2. Meningitis and encephalitis.
3. Familial degenerative diseases, e.g. Tay-Sachs' and amaurotic familial idiocy.
4. Cretinism.
5. Isolated sensory defects such as deafness and blindness.
6. Arthrogryposis multiplex congenita.
7. Amyotonia congenita.
8. Brain tumour.

REFERENCES

1. Gregg, N. McA. (1941): Trans. Ophthal. Soc. Austral., **3,** 35.
2. Swan, C. (1949): J. Obstet. Gynaec. Brit. Emp., **56,** 591.
3. Phelps, W. M. (1950): Cerebral Palsy Institute Proceedings. New York.
4. Pohl, J. F. (1950): *Cerebral Palsy.* St. Paul, Min.
5. Medalie, M. Personal communication.
6. Evans, E. S. (1946): Proc. Roy. Soc. Med., **34,** 317.
7. Collis, E. (1954): Arch. Dis. Childh., **29,** 113.

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EDITORIAL

BANNING A DRUG

The medical profession in Great Britain has been thrown into a state of great agitation over the British Government's intention to ban the manufacture of heroin (diacetylmorphine), which is generally accepted as the most dangerous drug of addiction known to man. Correspondence columns in the lay press have been filled with letters from all manner of persons—medical men and patients, addicts and laymen—both approving and decrying the Government's decision. No drug has ever been banned in Britain before, and many doctors—including an influential section of the British Medical Association—view this move as a real threat to medical freedom. A 'Fellowship for Freedom in Medicine' has taken up the cudgels for heroin and avowed that it will fight the Government on this point to the bitter end. Opposing this view are the official advisory committees of the Ministry of Health, comprising some of Britain's most eminent medical scientists and clinicians, who actually suggested the ban in the first place, after the World Health Organization had recommended it to its members in 1953.[1]

'Although it took a long time for the medical profession to realize the great danger of addiction to heroin, it was not long before the underworld and smugglers discovered its euphoric properties . . . . In 1924 it was estimated that, while only 58 ounces of heroin was prescribed by the entire medical profession in New York State, a total of 76,000 ounces was used by the narcotic addicts in New York City'.[1] The legitimate production ceased in USA after 1924, but it is still manufactured in other countries, from which it is available to smugglers. The WHO recommendation of 1953 was based on the fact that if heroin were not manufactured, even for legitimate purposes, it could not be used illicitly.

In South Africa the Union Government decided to act on the recommendation, and since 1952 permits to import heroin have not been granted (it is not manufactured in South Africa). In cough mixtures heroin has been replaced by other morphine derivatives such as codeine, ethyl morphine hydrochloride and, more recently, Pholcodine, which is reputed to be an even more efficacious cough repressant than heroin, and to be free from the excessive tendency of the latter to

VAN DIE REDAKSIE

'N MIDDEL IN DIE BAN

Die mediese beroep in Groot-Brittanje is baie ontsteld omdat die Britse regering besluit het om die vervaardiging van heroïen (diasetielmorfien) te verbied. Heroïen word algemeen beskou as die gevaarlikste gewoontevormende middel wat die mens ken. Die briewerubrieke in die lekepers is vol briewe van allerhande mense—dokters en pasiënte, verslaafdes en leke—briewe wat die regering se besluit goedkeur en briewe wat dit afkeur. Nog nooit tevore is 'n middel in Brittanje in die ban gedoen nie, en baie geneeshere, waaronder 'n invloedryke groep in die British Medical Association, beskou hierdie stap as 'n wesenlike bedreiging vir mediese vryheid. 'n ‚Fellowship for Freedom in Medicine' het in die bres getree vir heroïen, met die uitgesproke voorneme om die regering in hierdie saak tot bittereinde te bestry. Aan die ander kant staan die amptelike raadgewende komitees van die Ministerie van Gesondheid, behelsende party van Brittanje se mees vooraanstaande mediese wetenskaplikes en klinikusse, wat eintlik heeleerste die ban voorgestel het nadat die Wêreld-Gesondheidsorganisasie dit in 1953 by sy lede aanbeveel het.[1]

‚Although it took a long time for the medical profession to realize the great danger of addiction to heroin, it was not long before the underworld and smugglers discovered its euphoric properties . . . . In 1924 it was estimated that, while only 58 ounces of heroin was prescribed by the entire medical profession in New York State, a total of 76,000 ounces was used by the narcotic addicts in New York City'.[1] Sedert 1924 is die wettige produksie van heroïen in die V.S.A. gestaak, maar dit word nog in ander lande vervaardig waar smokkelaars dit kan bekom. Die aanbeveling wat die Wêrelds-Gesondheidsorganisasie in 1953 gemaak het was gebaseer op die feit dat indien heroïen selfs nie eers vir geregverdige doeleindes vervaardig word nie, dit ook nie onwettig gebruik kan word nie.

In Suid-Afrika het die regering besluit om volgens hierdie aanbeveling te handel, en sedert 1952 is geen permitte vir die invoer van heroïen toegestaan nie (dit word nie hier vervaardig nie). Die heroïen in hoesmiddels is vervang deur ander derivate van morfien, soos kodeïen, etielmorfienhidrochloried, en vandag ook Pholcodine. Laasgenoemde is na bewering 'n doeltreffender hoesonderdrukker as heroïen, en is baie minder gewoontevormend. Selfs voordat heroïen in Suid-Afrika uitgesluit is, het die mediese beroep al grotendeels opgehou om dit voor te skryf, en die ban

induce addiction. Even before heroin was excluded the medical profession in South Africa had already largely ceased to prescribe it, and the ban came into force with no resulting inconvenience and with none of the adverse reaction in the medical profession that it has produced in the UK.

In 1953 the British Pharmacopoeia Commission decided to omit heroin from the B.P., and by that time about 50 countries actively opposed its manufacture for therapeutic purposes. In the United States, where the drug was banned before the second world war, the view has long been held that heroin is completely replaceable in clinical practice. 'Heroin is such a dangerous drug that its use has been banned in America and it would be a great advantage if this control was exercised in all other countries', states a standard British text-book of pharmacology.[2] Its action is similar to that of morphine—'only more so'; it is a far greater depressor of the respiratory system, and its analgesic action is said to be 4 times that of morphine; and the risk of addiction is undoubtedly very great. In fact, 'heroin shares all the disadvantages of morphine without any serious advantages'.[3] Both as an analgesic and as a cough repressant it is ordinarily used as a last resort, but not invariably. Some practitioners swear by linctus of diamorphine *B.P.C.*, yet the modern view (and certainly the experience of the American profession) is undoubtedly that heroin is in no way superior to other drugs such as pethidine, methadone, etc. Pathetic descriptions in the lay press of the indispensability of heroin in cases of inoperable terminal carcinoma are hardly supported by the weight of responsible medical opinion. And as a cough repressant heroin cannot be held to stand alone.

There are, of course, two sides to the argument. On the one hand the *Lancet* has reiterated its previously-given advice to the Government, namely to fulfil its international commitments and do what the United Nations asks it to do:[4] on the other hand there is the attitude of standing on principle epitomized by Sir Reginald Watson-Jones's recent letter to the *Times:* 'If we are to ban every drug that might lead to addiction we should also ban alcohol and tobacco and we must wonder whether the Government are prepared to do this'[5]—not, it seems to us, a very convincing argument.

1. Editorial (1953): S. Afr. Med. J., **27,** 540.
2. Wilson, A. and Child, H. O. (1952): *Clark's Applied Pharmacology*, 8th ed., p. 267. London: Churchill.
3. Sollmann, T. (1944): *Manual of Pharmacology*, 6th ed., p. 288. Philadelphia: Saunders.
4. Editorial (1955): Lancet, **2,**
5. Letters to the Editor, *Times*, London, 3 December 1955.

het dus in werking getree sonder ongerief en sonder die vyandigheid wat dit onder die mediese beroep in die V.K. uitgelok het.

In 1953 het die Britse Pharmacopoea Commission besluit om heroïen uit die B.P. weg te laat, en teen daardie tyd was omtrent 50 lande al aktief gekant teen die vervaardiging van heroïen vir behandelingsdoeleindes. In die Verenigde State, waar hierdie middel alreeds voor die tweede wêreldsoorlog verbied is, is dit al lank die oorheersende mening dat heroïen heel maklik in die kliniek vervang kan word. ‚Heroin is such a dangerous drug that its use has been banned in America and it would be a great advantage if this control was exercised in all other countries', verklaar 'n Britse standaardwerk oor farmakologie.[2] Heroïen se werking kom ooreen met dié van morfien—maar dit is veel kragtiger; dit is 'n baie kragtiger onderdrukker van die asemhalingstelsel sy pynstillende aksie is 4 maal sterker as dié van morfien, en die gevaar vir verslawing is ongetwyfeld baie groot. Met reg kan dit gesê word dat ‚heroin shares all the disadvantages of morphine without any serious advantages'.[3] Beide as pynverdower en hoesverligter word dit gewoonlik as laaste redmiddel gegee, maar ook nie altyd nie. Sommige geneeshere het groot vertroue in linctus van diamorfien *B.P.C.*, maar die moderne beskouing (en gewis die ondervinding van die beroep in Amerika) is dat heroïen in geen opsig ander middels soos pethidine, methadone ens. oortref nie. Die lekepers se patetiese beskrywings van die onmisbaarheid van heroïen in gevalle van onopereerbare terminale karsinoom klop nouliks met die algemene en oorwoë mediese opinie. As hoesonderdrukker is heroïen nie enig nie.

Natuurlik het die strydvraag twee sye. Aan die een kant het die *Lancet* sy vorige raad aan die regering herhaal toe hy die regering aangeraai het om sy internasionale verpligtinge na te kom en die opdrag van die Verenigde Volkere uit te voer;[4] aan die ander kant is daar dié wat dit as 'n beginselsaak beskou, soos hul voorstander sir Reginald Watson-Jones wat onlangs as volg aan die *Times* geskryf het: ‚If we are to ban every drug that might lead to addiction we should also ban alcohol and tobacco and we must wonder whether the Government are prepared to do this'[5]—vir ons klink dit nie juis oortuigend nie.

1. Van die Redaksie (1953): S. Afr. T. Geneesk., **27,** 540.
2. Wilson, A. en Child, H. O. (1952): *Clark's Applied Pharmacology*, 8ste uitg., p. 267. London: Churchill.
3. Sollmann, T. (1944): *Manual of Pharmacology*, 6de uitg., p. 288. Philadelphia: Saunders.
4. Van die Redaksie (1955): Lancet, **2,**
5. Briewe aan die Redakteur, *Times*, Londen, 3 Desember 1955.

DEATH OF DR. PETER ALLAN

We regret to announce the death, which occurred suddenly at Hermanus on Tuesday, 10 January 1956, of Dr. Peter Allan, former Secretary for Health. He was 69.

Dr. Allan took his medical degree at the University of Edinburgh and came to South Africa in 1920. One of his first tasks was a Union-wide survey of tuberculosis. In 1923 he opened the first tuberculosis hospital in South Africa, the Nelspoort Sanatorium, and, apart from a break of 3 years as field research officer to the Tuberculosis Research Committee, spent the next ten years there.

An in memoriam notice of Dr. Allan will appear in a later edition of the *Journal.*

THE HEART, THE PHYSICIAN AND THE PSYCHIATRIST*

DAVID PERK, M.D., D.P.M.

Johannesburg

The heart draws the physician and psychiatrist together in a number of clinical situations, and an attempt is made in this paper to review them, with special reference to their psychogenesis, and to indicate what help the psychiatrist can bring to the relief of the clinical conditions under examination.

1. CARDIAC DISEASE AND PSYCHOLOGICAL REACTIONS

The psychological symptoms resulting from organic cardiac disease will depend on whether the cardiac condition comes on precipitately or insiduously, whether the condition is transitory and, if not, the degree of compensation evoked, and finally the pre-morbid personality. The psychological symptoms can be divided into (*a*) personality disturbance, and (*b*) symptoms resulting from physiological disturbance of the brain.

CORONARY THROMBOSIS

Generally speaking, the psychological reactions to a recent coronary thrombosis, and the symptoms they embrace, fall under the following headings: (1) anxiety reaction, (2) depression, (3) disturbance of body image, and (4) cerebral symptoms.

Anxiety Reaction

It is understandable that a degree of anxiety should accompany the occurrence of a coronary thrombosis. This psychological aspect of the condition may escape notice in the concentration on the physical condition. When the critical phase is over, and depending on the personality of the patient and on the way he is being managed by his doctor or doctors, a morbid anxiety may set in. The individual worries beyond cause and reason about his business, his finances, his family responsibilities, with the unspoken fear that he has little time if any to set his house in order. He becomes generally apprehensive, is afraid to be left alone, afraid of company, afraid to sleep. He lacks application, and feels restless and may become agitated. Somatic manifestations of anxiety occur—bouts of diarrhoea, excessive sweating, palpitations, giddiness, etc.

The development of an anxiety state in the course of a coronary thrombosis has an adverse influence on the cardiac condition. In a recent case I saw, the patient (Mr. K., aged 53) was so agitated that when every means of sedating him failed, ECT (electro-convulsive treatment) had to be applied. Despite the physician's warnings, he insisted on being motored 30 miles to his farm to reassure himself that the cattle were being properly tended. He was never reassured by these visits, however; and there can be little doubt that the anxiety about the cattle was essentially a displacement of anxiety about himself (see below). He was so afraid to go to sleep that he stayed awake far into the night, keeping the household awake with him, placed himself in an armchair for the night's sleep, waking almost hourly and rousing his wife to keep him company.

This patient kept on going into failure, and I have little doubt that the physician will attribute it to the excessive activity of the patient, on the one hand, subjecting the weakened heart to needless demands on it, and to the physical effect of anxiety on the other, mediated through the autonomic system. The ECT helped to allay the patient's agitation and at no time during this therapy—he had 10 treatments—did he go into failure. In the presence of myocardial infarction, both physician and psychiatrist are chary of employing ECT, but if the alternatives presented are a patient exhausting his cardiac reserve through anxiety and agitation or the physician exposing his patient to the risk of a catastrophe induced by ECT, then the latter is probably the lesser risk, unless the heart is in a very grave condition. I have never had a fatality in the use of ECT in damaged hearts. That is not to say, however, that there is no risk to heart and life. Where there is a clear psychiatric indication for its use, the contra-indication should, in my opinion, be only such a degree of myocardial damage that life is in the balance; and the clearest indication for ECT in coronary thrombosis is agitation. ECT is more often given for depression, and with the depressive reaction I shall deal later, but agitation may be present without any marked degree of depression, and is a bigger threat to the heart than depression.

In advising ECT to an anxious patient, the first response is usually a hostile one, born out of his anxiety; but as one remedy after another fails to give him rest and peace of mind, he is, sooner or later, likely to be won over to the idea. If proof were wanted that anxiety is harmful to the heart and helps to precipitate cardiac failure in the damaged heart, it is available in the observation that when the patient relapses mentally, after cessation of ECT, a physical relapse follows shortly after.

I have mentioned that Mr. K. was afraid to go to sleep. This is, of course, an inevitable sequel of disturbed sleep, and is unfailingly present with severe anxiety. The anxious preoccupations of the day are magnified at night and inflamed by the anxieties buried within the unconscious. The result is that the patient keeps waking, feeling uneasy, worried or panicky, and this further aggravates the noctural tendency to cardiac asthma. The effect of these nightly experiences is to aggravate the fear of death, already aroused by the heart condition, and makes the patient fight with and chase away sleep, as if he were fighting for his very life. The patient in such a plight demands stronger and stronger hypnotics with little benefit or satisfaction. The danger of introducing habit-forming drugs to such

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a patient must be obvious to all, and the better remedy, I suggest, is to give ECT.

The human capacity for enduring anguish has its limitations. When the physical integrity of the ego is threatened objective appraisal and contemplation of the threat is, as a rule, rendered well-nigh impossible by various devices which are unconsciously stimulated. One of these is displacement of anxiety (see above). The anxiety aroused by the recognition that life is threatened is repressed and unconsciously displaced onto other causes of anxiety, real or imagined, but external to the ego. Mr. K. worried about his cattle. A medical man I saw recently began to worry about the arrangement he had concluded with his assistant to carry on his practice during his illness. When one questions the subject of a recent coronary thrombosis about his anxieties, the one he does not mention, because he is not in fact fully conscious of it, is his anxiety about himself. He confides, however, other anxieties which preoccupy him and are, to a greater or lesser extent, absorbing the primary anxiety about his condition.

Some of the somatic symptoms complained of by patients are probably not directly related to the cardiac lesion, but are referable to the physiological disturbance consequent on the anxiety, as, for example, peripheral paraesthesiae and abdominal paraesthesiae, such as emptiness or coldness in the abdomen.

Disturbance of Body Image

There is a disturbance of the body image in cases of coronary thrombosis which, I imagine, few physicians will have failed to observe; and that is the total denial or refusal to recognize that the heart has suffered damage. Either the patient insists he is all right or wants to carry on his life as if nothing has happened. I had one patient who wanted to carry on with a previously planned trip overseas, and another who could not be persuaded to give up his mountaineering, not to mention other patients who insist on returning to active business almost immediately. Nielsen (1948) has referred to this non-recognition of a pathological state as a disturbance of the physiological body-image.

The converse to the above picture is presented by the patient with hypochondriacal preoccupation. Here the disturbed body-image forms the nidus for a number of mechanisms operating within the psyche. One is the tendency to hypochondriacal rumination. Some individuals are more disposed than others to keep track of their bodily sensations and are, of course, more likely to be come absorbed in them when they undergo pathological change. One has heard it alleged that the injudicious address of the medical attendant to his patient and his fussy management of the case may be responsible for his patient's hypochondriasis. I believe the fault lies essentially with the patient's premorbid personality. This does not, of course, refer to iatrogenic cardiac neurosis, with which I shall deal later.

When the critical phase is over, and as the period of convalescence lengthens, the hypochondriacal individual becomes progressively more preoccupied with his condition and its daily state. He becomes obsessive in his scrupulous observation of the restrictions imposed on him by his doctors, exceeds them and becomes panicky when he is pressed to relax them. He wants reassurance and has no faith in it, and slowly, or perhaps not so slowly, settles down to the life of an invalid, refusing to test his cardiac capacity, narrowing down his interests progressively until his waking hours are spent in keeping an inventory of his aches and pains and incapacities, and taking medicines because it is part of the day's ritual and not because he has belief in them.

A modification of the above is where there is less obsessiveness and more anxiety accompanying the hypochondriacal preoccupation.

Another mechanism is that of delusion formation, where the affected organ becomes the subject of hypochondriacal delusions. I cannot say that I have seen this type of psychotic reaction in cases of coronary thrombosis, though I have seen coronary thrombosis occurring in patients with hypochondriacal delusions which embrace the heart as well as other organs.

Depression

A depressive reaction occurs not uncommonly in association with coronary thrombosis, with the usual features of hopelessness, purposelessness, worthlessness and retardation. Where the depressive reaction has succeeded a period of anxiety and agitation the condition fluctuates between intervals of anxiety and agitation and depression, and in the latter it arouses suicidal reflections and impulses. Delusions of financial ruin and guilt are expressed. The more the depression reflects the precipitation of an endogenous depression the more abundant and bizarre the delusions, and the hypochondriacal delusions especially point to a severe degree of depression which is likely to be endogenous in character.

Early-morning wakefulness in a depressed mood which is at its worst at that time is particularly significant of the transition from an anxiety reaction to a depressive reaction.

I have mentioned the use of ECT in cases of anxiety and agitation; the indication for its employment is even clearer in cases of depression associated with coronary thrombosis, and the benefit is likely to be greater and more lasting to the extent that the depressive reaction is unassociated with anxiety. Unless one bears this in mind, disappointment may be felt that the relief obtained from ECT is not fully sustained. As anxiety is present in most of the psychiatric reactions to coronary thrombosis, the aid to be obtained from ECT may be found to be not fully lasting; but it can be life-saving, and in any case even temporary relief is welcome to the patient. The relapse which may follow the cessation of treatment, after a variable interval, does not, in any case, bring back the patient to the clinical level which existed before treatment.

Cerebral Symptoms

The reactions mentioned so far are essentially psychical in character. That is not to say that the cerebral anaemia which results from coronary thrombosis has no part in precipitating or sustaining these reactions, but that they are basically affective responses to an ego-threat. There are, in addition, symptoms which directly

reflect the cerebral anaemia consequent on coronary thrombosis, such as the loss of consciousness or epileptiform convulsions that may accompany the occurrence of coronary thrombosis, or the confusion and intellectual impairment which may be present for a variable interval after the event.

Aetiology of Coronary Thrombosis

In the search for the aetiology of coronary thrombosis the personality of its victims has come under suspicion. Generally speaking, they are people who take their responsibilities very seriously, and set themselves high standards of achievement. Their make-up conspires with circumstances to establish this disposition. The effect is that they are continually driving themselves, fearful and eventually unable to relax, and anxious with the least obstacle or threat to their self-set programmes. Their interest and energies have one focus and goal, success and security, with the result that they live in the narrow groove of effort, tense and earnest and often humourless. Their mission is a closely guarded secret and they are equally reticent about their anxieties. At what point the coronary attack occurs, and its relationship to the special stress of circumstances, it is difficult to dogmatize about. Physical factors no doubt play their part, very probably the major part, in predisposing the individual to an attack, and in precipitating it. Nevertheless, it is a common observation that financial stress and catastrophe march hand in hand with coronary thrombosis.

OTHER CARDIAC LESIONS

The psychiatric reactions that are associated with other cardiac lesions are divisible, like those connected with coronary thrombosis, into two classes; the essentially psychical reactions and essentially organic reactions. To the extent that a cardiac lesion reduces the cerebral blood-supply the resulting psychiatric symptoms are more likely to be of the organic-reaction type than of the non-organic type. That is not to say that cerebral anaemia is not an operative mechanism even when the reaction appears to be essentially psychical, but as the symptoms come to take on more of the appearance of an organic reaction the casual responsibility must lie more with cerebral anaemia.

The organic symptoms are of 3 degrees—acute, subacute and chronic—depending on whether the cardiac lesion has arisen suddenly or insiduously. In addition, and this applies especially to the more chronic cardiac lesions, the symptoms derive in part from the causal lesion and in part from the resulting lesion; as, for example, hypertension and the ensuing cardiac failure. In yet another class of case, that of arteriosclerosis where the arterial changes affect both the coronary and cerebral vessels, the symptoms are based on a cerebral anaemia for which both heart and cerebral vessels are responsible. In arteriosclerotic dementia the heart hardly ever escapes from the arteriosclerotic process.

Marchand (1955) recently made an interesting observation on the occurrence of myocardial infarction in psychotic patients. He said that pain was absent in 82·5% of cases, in sharp contrast to the much lower frequency of painless myocardial infarction in the general population. He gave it as his opinion that the explanation lay in the fact that the meaning of pain was lost to the majority of patients with psychoses, both organic and functional.

Peskin (1955) has recorded a case, which I had the opportunity of examining with him, which cardiologically presented a combination of mitral stenosis and a 3 : 1 flutter and, from the psychiatric aspect, 'blackouts' and depression. As happens so frequently in psychiatric conditions, there were a number of aetiological factors in operation, and so little was the heart condition suspected of responsibility for the 'blackouts' and the depression that the former was suspected of being hysterical and the latter endogenous. He was in fact referred to Tara Hospital for shock treatment. A valvotomy did little to change the psychiatric picture, but after non-convulsive cerebral electro-stimulation a normal cardiac rhythm was re-established, and with it the 'blackouts' and depression disappeared. This case well illustrates the direct relationship between cardiac lesions, cerebral anaemia and psychiatric symptoms. It also brings to light a new form of treatment for cardiac arrhythmias. Lewis, Richardson and Gahagan (1955), on the other hand, found that nearly 80% of the series of 21 patients with pathological cardiovascular conditions that they treated with electric stimulation developed cardiac arrhythmias of various degrees of severity. Because of the risks involved in the treatment, they gave it under continuous electrocardiography and, in addition to the usual precautionary measures of pre-treatment fast of 3 hours, rapid and short-acting intravenous anaesthesia and scoline, they gave intravenous atropine to block the cardiac portion of the vagus nerve, and either quinidine or chlorpromazine to reduce the treatment-induced extravagal arrhythmias.

With heart failure coming on insidiously the patient may complain of lack of concentration, undue mental fatiguability, insomnia, emotional lability, impoverishment of memory. Anxiety, agitation, depressive and paranoid reactions may occur, and dementia may develop. Mayer-Gross, Slater and Roth (1954) mention that hypomanic pictures and delirious states may occur.

Prognosis

The prognosis in the psychiatric reactions to cardiac lesions depends on two considerations in the main. First, to the extent that it is based on cardiac pathology, the condition will run parallel to it. Secondly, the endogenous component provoked by the cardiac lesion will run a course that is determined largely by the dynamics that are inherent in it. Roughly speaking, therefore, the more profound the psychiatric disturbance aroused by the cardiac lesion the longer is it likely to endure beyond the time-span of the cardiac lesion itself. Caution is required in predicting the likely progress of a psychiatric reaction to a cardiac lesion that does not quickly clear up, both because it is not easy to assess the unconscious difficulties that have been unleashed nor, I suppose, for the physician, to estimate the danger of recurrence of the cardiac lesion.

An acute confusion developing in the course of chronic heart failure is likely to be a terminal phenomenon.

Diagnosis

The diagnosis of the psychiatric reaction, as a descriptive entity, presents little difficulty. The aetiological diagnosis can only be made, with confidence, with the efflux of time and the progress of the cardiac lesion.

Two special circumstances should put the psychiatrist on his guard when he comes to making a diagnosis, and bid him keep in mind the possibility of a coronary thrombosis. One is met with in dealing with an unconscious patient, of middle age and beyond, when what may have the appearance of a cerebral apoplexy may be coronary infarction. The other, which is not infrequently met with in mental hospitals, is a sudden change in the behaviour of a psychotic patient, as, for instance, in the case of the patient who has slavishly and energetically applied himself to certain routine jobs in the hospital and who suddenly prefers to stay in bed, or to remain in the ward instead of going into the farmlands; electrocardiography in such instances would reveal not a few cases of coronary thrombosis (Marchand 1955).

II. Cardiac Symptoms in a Neurotic Setting in the Absence of Cardiac Disease

EFFORT SYNDROME

Symptoms referable to the cardiovascular system are present at some time or another in most cases of anxiety neurosis; yet in civilian life the psychiatrist very uncommonly refers to such cases as effort syndrome. In World War I, however, about 60,000 cases with such symptoms had been diagnosed as effort syndrome in the British Army by the summer of 1918 (Lewis 1940). It was Thomas Lewis who separated this condition from organic cardiovascular disorder, and though he appreciated that there was a psychological factor in operation he believed the condition to be a definite clinical entity. At the outbreak of World War II, in anticipation of the recurrence of the syndrome in war time, an effort-syndrome unit was established within the E.M.S. (emergency medical service) in England, and in the Middle East the British Army set up two effort-syndrome centres. It was the work done in Mill Hill, London, that demonstrated that neither aetiologically nor symptomatologically was there any valid ground for applying a special label to symptoms which were psychiatric in origin and were to be found in a number of different psychiatric conditions. Jones and Lewis (1941) found 100 successive admissions of effort syndrome to be classifiable as follows: anxiety state, acute and chronic, 31%; depression 12%; psychopathic personality 18%; hysteria 11%; post-infective 11%; hyperventilation tetany 1%; organic disease 11%; high-grade mental defect 4%; schizophrenia 1%. Early in the war High Authority requested that the label 'effort syndrome' should be avoided, with the result that the two effort syndrome centres in the Middle East faded out, and effort syndrome became a relic of the past.

The elimination of effort syndrome as a label may not have made any real difference to the number of casualties in the war. Nevertheless, it prevented harmful hospitalization and the creation of neuroticism by suggestion.

In World War I 5/6ths of the sick cases classed as cardiovascular were effort syndrome. In civilian life, according to Paul Wood (1950), 'approximately 1 in 3 or 1 in 4 patients who present themselves at cardiovascular clinics are suffering from psychoneurosis without organic disease of the heart'. It is possible therefore that the elimination of the label 'effort syndrome' may have obscured an important fact, namely, that in wartime more symptoms presented by the psychoneurotic patients are referred to the cardiovascular system than in peace time. The inference to be drawn from this is that the special circumstances of war provoke a psychoneurotic response which obtains most dramatic expression *via* cardiac symptoms. In other words, it is in special measure a war neurosis, and it may be that the Mill Hill team failed to see this angle, when they sought and produced support, in clinical observations and figures, for their view that there was no warrant for the use of the term 'effort syndrome'. Effort syndrome it certainly was not, because the reaction could obtain when the subject was at rest and, again, the affected soldier could engage in some energetic activity in which he was interested without getting the symptoms. On the other hand, it was a reaction which obtained prominence during war time and, though there was no constant causal environmental factor to be found in the cases, they all had this in common, that they occurred against the background of war and its multitudinous stresses of fear and of assault on established habits, conventions and dignities. I do not, however, want to labour this question of terminology. What I am anxious to do is to see what light the wartime cases of effort syndrome can throw on the subject of cardiac neurosis, and in particular on the questions: (*a*) What types of personality develops cardiac neurosis, (*b*) the circumstances that precipitate it, and (*c*) why the heart is chosen as a setting for staging the drama of the battle between the psyche and the external world.

There appear to be 3 predisposing factors, viz. (*a*) a family history of neurosis, psychopathy, insanity and epilepsy, (*b*) physical inferiority, and (*c*) a particular kind of psychical make-up.

Family History. In a series of 85 cases that Paul Wood (1941) investigated, he found a positive family-history in about 56%, and a cardiac neurosis was present in about 50% of these. These figures indicate how the individual is prepared by home training for his later maladjusted reactions.

Physical Inferiority. Wittkower and Rodger (1941) found that 15 of their series of 50 gave evidence of life-long physical inferiority.

Psychical Make-up. Wittkower and Rodger (1941) make the following comments: 'None of the patients suffering from effort syndrome was found to be emotionally well-adjusted. The vast majority conformed to the obsessional type, possessing an over-keen sense of duty and over-rigid morality'. 'The effort-syndrome patient is preoccupied with problems of morality, self-regard,

religiosity, patriotism, duty, responsibility and self-sacrifice'. Needless to say, this make-up had made itself manifest in various ways long before the illness of the effort syndrome. Thus Jones and Lewis (1941) found that 50% of their patients 'had had, often for the greater part of their lives, symptoms of vegetative disorder (such as breathlessness, palpitation, excessive sweating or diarrhoea) on slight provocation of an emotional sort', '40% of them had not played any games, in many instances, because it upset them'; half had shown neurotic traits, like stammering, bed-wetting, sleep-walking; 'nearly 2/3rds had shown more or less pronounced anomaly of personality—shy, tense, hypochondriacal, sensitive or delinquent'. '67% of the patients had had symptoms of effort syndrome before the war', in many dating from their early school days. Paul Wood (1950) found that 'the most constant characterstic is timidity, which results in lack of confidence, a sense of inferiority and undue apprehension. On such a soil an anxiety state takes root very easily'.

There are two clinical aspects to effort syndrome; the psychic and the psychosomatic. As just indicated, there are a number of psychiatric conditions found associated with the somatic symptoms of effort syndrome. There would thus appear to be two levels of personality predisposition, the one predisposing the individual to a personality disturbance with its specific features, and the other determining reactively for the individual concerned, in both the aetiological and psycho-mechanistic sense, the genesis of psychosomatic symptoms in response to a stressful or traumatic situation.

What determines the somatic manifestations of the effort syndrome? It was mentioned earlier that in 50% of cases there was a family history of cardiac neurosis. This means that the body image borne by these individuals includes a sub-image of dysfunction in certain circumstances. This sub-image is supplemented by further sub-images that are based on the fears and anticipations of difficulty and defeat aroused by any physical defects or inadequacy that may be present or imagined. Because of timidity, for instance, the individual may feel that he is physically unequal to a particular test of physical strength or endurance, and when he is confronted with such a test he reacts with the dysfunction previously rehearsed mentally.

At a deeper level it is probable that complexes are represented in the body image by physiological correlates. Thus the breathlessness of the effort syndrome might be a reflection of the fear of suffocation that lies at the heart of claustrophobia (Wittkower and Rodger 1941).

In addition to the symptoms that are latent in the body image, others derive from the stimulation or inhibition of the vegetative system by the emotion aroused in a particular situation as, for example, anxiety or depression. How far the body image determines the vegetative expression of emotion and how it is reflexly stimulated by peripheral somatic experience, is a subject that would take me too far afield.

Predisposition, divided into factors, creates the appearance of something formal and static residing within the mind, or perhaps a spring set for action but inactive until it is touched off. But it is more than that. It is also something dynamic, a ferment of conflicts and strivings which keeps changing the formal predisposition and the susceptibilities it imposes on the individual, and shapes the reaction that results from the clash with the environment. The precordial pain of the effort syndrome case may, in the first instance, reflect and represent guilt over aggressive drives and the self-punishment attendant on it, and secondarily, may be unconsciously sustained by the necessity to escape from the painful conflict.

Predisposition, psychical and psycho-physiological, is a pathological mechanism that is evolved under the impact of various influences, from early life, to preserve the infantile ego and, when circumstances arise that challenge this narcissistic ego, the effort syndrome, amongst other reactions, emerges or erupts, according to the violence of the challenge. It is worthy of note that the effort syndrome occurred in relatively few cases of battle exhaustion (1-2%), whereas in non-combat psychoneurosis it occurred in about 20% of cases.

Clinical Features. According to Paul Wood (1950) the chief symptoms are breathlessness, palpitations, fatigue, left inframammary pain, dizziness, excessive sweating and headache; and the most important signs are nervousness and depression, tremor, frequent sighs or hyperventilation, clammy cold hands, tachycardia, raised casual blood-pressure around 160/90 mm. Hg, and inability to hold the breath for longer than 20 seconds.

Diagnosis. The combination of psychiatric and psychosomatic symptoms as a reaction of maladjustment to a situation points unmistakably to the diagnosis. The recognition that it was a psycho-reactive entity led to the abolition of the term 'effort syndrome' in World War II and its replacement by labels descriptive of the psychiatric presentation. In peacetime the effort-syndrome case is apt to be labelled hyperventilation syndrome by the physician, and anxiety state or anxiety-hysteria by the psychiatrist.

Psycho-physiological Mechanism. According to Paul Wood (1941) it was Soley and Shock (1938) who first claimed that hyperventilation was responsible for the symptoms of effort syndrome. He reviewed the evidence for the claim and gave it as his considered opinion that when present, and it was present in only 21·5% of his series of 200 cases, hyperventilation 'must aggravate certain of the symptoms. In any case, spontaneous hyperventilation is not a diagnosis, but apart from rare organic causes, merely one mechanism by which psychosomatic manifestations are produced'. There are, of course, other physiological mechanisms for translating emotion into somatic symptoms, which operate more universally. I refer to the action of the pituitary and adrenals, and possibly other endocrine glands, which affect the soma directly or *via* the vegetative system or the central nervous system, and the action of the thalamus and hypothalamus, which affect the soma *via* the central nervous system or the autonomic system or the pituitary.

Whatever the route, there is evidence (Dunbar quoting Wolf, 1948) that psychical influences can produce alterations in the rate and size of the heart and, in the

electrocardiogram, that anxiety can produce coronary spasm. It is also believed (*Ibid.*) that coronary spasm or occlusion produces anxiety as a direct response, in the same way that a peptic ulcer, for example, produces pain. The suggestion therefore is that anxiety can produce coronary disease and coronary disease anxiety. Nevertheless, though anxiety can produce coronary disease, there are instances as, for example, in effort syndrome, where anxiety produces symptoms that stimulate coronary disease and yet do not provoke any overt organic change in the coronaries. What is the factor or factors that determine whether anxiety is to produce a cardiac neurosis or a coronary disease? It may be based on a physical factor or factors which are unevenly distributed in the population, or on a personality factor. It is my belief that the predisposed individual who can verbalize his anxiety is likely to develop a cardiac neurosis, whilst the person whose capacity for emotional expression is more inhibited is likely to develop a coronary disease.

Prognosis. The patient with praecordial pain defies persuasion that it has nothing to do with his heart. Today, with the prevalance of coronary thrombosis, his greatest fear is represented by that condition. I have never come across a case of effort syndrome that has developed coronary thrombosis. Grant (1925) in a follow-up study of 601 men who had been discharged from the army in World War I with the diagnosis of effort syndrome, did not find a single case of coronary disease at the end of 5 years after discharge, and not a single case of death on account of coronary disease in the 52 deaths that had occurred.

In Paul Wood's (1941) series of 182 cases 31·1% returned to full duty, 36·3% returned to light duty, and 32·3% were discharged. In Grant's (1925) follow-up series, after 5 years 15·3% had recovered, 17·8% had improved and 56·2% were unchanged. The poor prognosis of cases discharged on account of effort syndrome is in line with the pronounced predisposition that operates in these cases.

III. Iatrogenic Cardiac Neurosis

The number of people who have been led to believe by a medical man that they have a diseased heart, through a misinterpretation of some innocent clinical sign as, for example, a soft systolic murmur at the apex, or by an injudicious comment to a patient regarding his heart, must be legion. The longer they live with that belief and order their daily lives to conform with it, the more difficult it is to shake it. It is extraordinary how often a person accepts the diagnosis of a diseased heart, and the shackles that go with it, without overmuch questioning or resistance. It rather suggests that the person unconsciously desires and seeks a defence through incapacity. However that may be, he certainly resists giving it up. It will be readily appreciated that such a subject is a candidate for the effort syndrome. The more integrated the personality the easier will it be to reassure the individual and persuade him to cast aside the illness that his medical attendant has unwittingly and unintentionally brought to him.

SUMMARY

Two psychiatric situations are associated with the heart, the one connected with organic heart disease and the other occurring in its absence. In the former the symptoms, which may be psychoneurotic or psychotic, are precipitated by the anxiety aroused or by cerebral anaemia, or both. Predisposition in the premorbid personality is a contributary factor. ECT is advocated for agitation and depression associated with coronary thrombosis; it can be life-saving.

Symptoms referred to the heart, unassociated with organic disease, are often described as effort syndrome. They are provoked by suggestion, either from the traumatic environmental situation or from an injudicious medical comment. Predisposition plays an essential part in the psychogenesis. A family history of cardiac neurosis is common, and the individual is highly susceptible to any threat to the narcissistic ego, to which he reacts with suppressed aggression.

Anxiety may cause coronary spasm and ultimately disease, but the effort syndrome case never goes on to coronary disease. Apart from physical factors the psychical make-up may determine whether anxiety ends in coronary disease or effort syndrome.

REFERENCES

Dunbar, F. (1948): *Synopsis of Psychosomatic Diagnosis and Treatment.* London:
Grant, R. J. (1925): Heart, **12,** 121.
Jones, M. and Lewis, A. (1941): Lancet, **1,** 813.
Lewis, W. H., Richardson, D. J. and Gahagan, L. H. (1955): New Engl. J. Med., **252,** 1016.
Lewis, T. (1940): *The Soldier's Heart and the Effort Syndrome.* London.
Marchand, W. E. (1955): New Engl. J. Med., **253,** 51.
Mayer-Gross, W., Slater, E. and Roth, M. (1954): *Clinical Psychiatry.* London.
Nielsen, J. M. (1948): *Agnosia, Apraxia, Aphasia. Their value in Cerebral Localization.* New York and London.
Peskin, M. (1955): Med. Proc., **1,** 99.
Soley, M. H. and Shock, N. W. (1938): Amer. J. Med. Sci., **196,** 840.
Wittkower, E. and Rodger, T. F. (1941): Lancet, **1,** 531.
Wood, P. (1941): Brit. Med. J., **1,** 767, 805, 845.
Idem. (1950): *Ibid.*, **2,** 33.

UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 28 December 1955.

Plague: Nil.

Smallpox, Transvaal: One (1) European case in the Nelspruit district.

Typhus Fever, Natal: Two (2) European cases in the Port Shepstone municipal area. Diagnosis confirmed by laboratory tests. *Orange Free State:* One (1) Native case in the Thaba 'Nchu district. Diagnosis confirmed by laboratory tests. *Cape Province:* No further cases have been reported from the Glen Grey district since the notification of 1 December 1955. This area may now be regarded as free from infection.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Chalna, Dacca (Pakistan).

Smallpox in Herat, Kabul, Kandahar (Afghanistan); Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Allahabad, Delhi (India); Dacca, Lahore (Pakistan).

Typhus Fever in Cairo (Egypt).

THE SURGICAL TREATMENT OF CONSTRICTIVE PERICARDITIS*

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The surgeon will be faced with conflicting and even contradictory views on the surgical treatment of constrictive pericarditis. It is my hope that this article, based on personal experience of thirty patients, will help to clarify some of the difficulties.

A knowledge of the anatomical and physiological changes and the pathology of constrictive pericarditis is a necessary prerequisite to an understanding of the problems which arise in the treatment of this disease.

ANATOMY AND PATHOLOGY

The pericardium consists of a fibrous layer and a serous layer which is reflected on to the myocardium as the epicardium. Normally the small quantity of fluid present in this serous cavity allows of free movement of the heart within it. The fibrous pericardium, which is attached to the diaphragm below and to the great vessels close to their entrance and exit from the heart, is also in close relationship to the parietal pleurae on the sides. The phrenic nerves and the pericardiaco-phrenic vessels run between the fibrous pericardium and the layers of the mediastinal pleura.

A right and a left lymph trunk drain the heart, the right trunk passing to the anterior mediastinal glands, while the left trunk passes to the tracheo-bronchial glands (Shore, 1929).

Excess fluid will collect posteriorly and to the left side, because the close proximity to the heart of the sternum in front and of the liver on the right and below precludes pericardial expansion in this area.

The pericardium is important in that it allows the heart to move freely in the thoracic cavity. The heart hangs almost as a suspended organ and moves freely in the pericardial cavity as the diaphragm moves downwards on inspiration. The pericardium also helps to prevent any sudden over-distension of the heart with blood.

The pathological changes in the pericardium may best be considered in reference to various stages, which may or may not terminate in constrictive pericarditis.

1. *Pericarditis.* This is the stage of acute infection and may be associated with gross constitutional changes. All the layers of the pericardium are involved, and tubercles may be present on the surface or on the serous layers.

2. *Pericarditis with Effusion.* The quantity of fluid may gradually increase until it reaches as much as 1,500 c.c. The effusion is usually clear. The pericardium becomes thickened and the serous surface loses its normal glossy lustre. At this stage some granulation tissue is present. The heart is still able to move freely within the sac, though its action is embarrassed by the presence of the effusion. The pear-shaped pericardium will accommodate itself to an increase in the quantity of pericardial fluid by enlarging until it occupies most of the lower half of the thoracic cavity. The heart continues to hang suspended in the pericardial fluid. The heart movements will be free, although the cardiac function will be impeded by the effects of tamponade. Resolution and return to normal can occur at this stage, so that no sign may remain of the pericarditis and the cardiac function may be unimpaired.

3. *Constrictive changes* constitute a later stage and may actually develop in the presence of a considerable pericardial effusion. Difficulty may be encountered in deciding whether the signs are those due to compression by the effusion or whether early constrictive changes have commenced. The term 'restriction' has been applied to the early stage of 'constriction', for which it is a pseudonym. In constriction the heart is encased in a close-fitting pericardium; active disease may still be present or the whole condition may settle down. The full effects of constriction are seen when further changes occur and the fibrous pericardium may be as much as 1 inch thick.

4. *Calcification.* Lamellae of fibrous tissue are deposited in the diseased pericardium and fibrotic areas interspersed with zones of active tuberculous granulation tissue are seen. Calcium is deposited in plaques, which may be thick enough to completely enclose the constricted heart. The tuberculous granulation tissue may dip deep into the myocardium, and small lacunae or lakes of pus may be shut off in the obliterated pericardial sac.

Two stages can be recognized clinically. The first stage is due to the tamponade effects of the effusion, while the second is due to the constriction effects of the fibrous adherent pericardium. The stages leading to constrictive pericarditis may take many years though, in several of the cases considered in this paper, the changes from pressure effects of effusion to the constrictive effects occurred in as short a period as 6 weeks. The symptoms were relieved by pericardial aspirations and yet a well-developed constrictive pericarditis was found at operation six weeks later.

Physiology

The mechanical effect of cardiac tamponade is to reduce the output of both the left and the right side of the heart. A similar effect is seen with the changes of constrictive pericarditis.

Left ventricular failure is a late complication. The left ventricle is able to pass on all the blood it receives and the real trouble lies in the fact that it actually does not receive enough blood. This explains why pulmonary oedema occurs as a complication of constrictive pericarditis. In spite of the increase in pulmonary-artery capillary pressure and the high diastolic pressure in the right ventricle, the left ventricle will still eject all that it receives. Even so, pulmonary arteriosclerosis is likely

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to result from the prolonged effect of tamponade or constriction. The right ventricle ejects all it can, but the outflow from the lungs has become obstructed by the constricted left ventricle. Any sudden increase in the amount of blood ejected from the right ventricle will increase the pressures in the pulmonary circulation, if the outflow from the left ventricle remains unchanged. An improvement in the ability of the left ventricle to increase its output without a corresponding increase from the right side, would be valueless.

The facts indicate that the left ventricle is the controlling factor in determining the pressure of blood in the pulmonary circulation, the right ventricle is responsible for the pressure in the venous system, and both ventricles together are responsible for the cardiac output. Failure of the left ventricle is a serious omen.

It is not easy to understand why so few cases of tuberculous constrictive pericarditis have been reported in South Africa. Clearly many cases of tuberculous pericarditis must occur, for the incidence of pulmonary tuberculosis in the Coloured and African peoples is the highest in the world. It is likely that many cases of

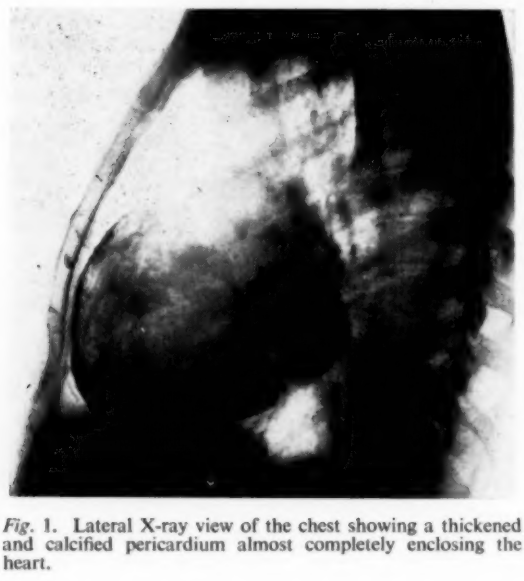

Fig. 1. Lateral X-ray view of the chest showing a thickened and calcified pericardium almost completely enclosing the heart.

constrictive pericarditis go unrecognized, because the degree of constriction may be so slight that the radiographic appearances pass as normal, unless there is associated calcification. The calcification itself may not be recognized unless special lateral views of the chest are taken. (Fig. 1). All cases of pulmonary tuberculosis should be examined for abnormal rise in the jugular venous pressure.

Constrictive pericarditis is not generally accepted as being due to tuberculosis. Evans and Jackson (1952), in view of the preponderant incidence of constrictive pericarditis in males, and as females are more commonly affected by rheumatic heart-disease, consider that constrictive pericarditis is the outcome of a tuberculous infection. Several authors have found causes other than tuberculosis and Bailey (1953) believes that constrictive pericarditis develops in a certain percentage (perhaps up to 5%) of all patients subjected to intra-pericardiac surgery unless the pericardial sac is left widely open. As the cause cannot always be clearly established, all cases should be regarded as tuberculous and should be given antituberculotic treatment prior to pericardiectomy. It is not easy to distinguish certain cases of nutritional heart-disease from tuberculous pericarditis.

INDICATIONS FOR PERICARDIECTOMY

When constriction has already occurred there will be no controversy about doing a decortication operation. The difficulty, as previously indicated, is to decide whether the compression of the heart is due to effusion or to a commencing constriction. Holman and Willett (1951) suggest a rule that as soon as a diagnosis of cardiac compression is made (in a case of presumed tuberculous pericarditis) an operation for cardiac decompression should be considered, regardless of whether the case is in the stage of pericardial effusion or in the stage of pericardial constriction. Our experience of cases presenting with pericardial effusion and signs of cardiac compression leads us to think otherwise. Many cases have responded to pericardial aspiration and treatment with antituberculotic drugs. Repeated aspirations may be necessary.

Routine treatment for pericarditis with effusion should be decompression, antibiotic treatment and careful observation. Disappearance of pericardial effusion with return of normal cardiac pulsation and normal jugular venous pressures has often justified this line of therapy. The persistence of high jugular venous pressure with a relatively immobile cardiac silhouette on screening indicates that constriction has developed and the operation of decortication is indicated. The actual constriction may take years to develop, but signs of increasing jugular venous pressure, in spite of pericardial aspirations and antibiotic treatment, would constitute a clear indication to proceed with decortication.

Though decompression may be an emergency procedure in cases of pericardial effusion, it is rarely urgent when constrictive changes have occurred. The patient has accommodated himself up to a point over a period of time. Improvement in the condition of the patient with heart failure has often been secured by means of bed rest, pleural and peritoneal aspirations, diuretics, digitalis and antibiotics. Several cases have shown so much improvement on these general lines that the patient has refused the pericardiectomy. The inevitability of the operation is only accepted by these patients when cardiac compensation has broken down once more.

Pericardiectomy should be performed when the indications of constriction are definite, even in the presence of active pulmonary tuberculosis.

The signs that the pericarditis has become of a constrictive type, so that pericardiectomy is indicated, are those of raised venous pressure, e.g. engorged veins in the neck, pulsation of engorged veins, and enlargement of the liver. The pulse rate is usually increased and the blood pressure low. Although there is a rapid circulation

time, the cardiac output is diminished. Minimal movement of the cardiac silhouette is seen on fluoroscopy. The size of the heart shadow is not necessarily diminished, because the pericardium is often grossly thickened. The superior vena-caval shadow may be enlarged. The ECG is usually of low voltage, the T-wave is usually low or inverted, and the P-wave may be notched.

PROCEDURE OF PERICARDIECTOMY

The Ventricles

Most authorities agree that both ventricles should be decorticated. Nevertheless, in two cases in the series now reported the left ventricles were decorticated to the satisfaction of the surgeon, but it was impossible to do the decortication of the right ventricles because the patients' general condition precluded further surgery; yet the ultimate clinical result surpassed all expectations, the patients being much improved and able to live comparatively normal lives.

Most surgeons commence with the decortication of the left ventricle. Schmieden *et al.* (1926, 1937) emphasized the importance of complete liberation of the left ventricle and considered it to be the only necessary surgical measure in most cases. Blalock and Burwell (1941) place major emphasis on the decortication of the right ventricle. Sellors (1946) states that both ventricles should be decorticated when possible. Decker (1947), influenced by the fact that the pericardium is a necessary organ, considers it sufficient to decorticate the left ventricle only. Holman and Willett (1949) emphasize the importance of a wide removal of diseased pericardium.

Constrictive pericarditis confines the two ventricles, and in my opinion both ventricles should be decorticated in order to permit of as normal a range of movement as possible. The left side should always be decorticated first in case of sudden engorgement of the lungs during the operation. This appears a rational physiological approach in spite of Klinkenbergh's statement (1951) that he first decorticates the right ventricle, without any fear of heart failure.

The Auricles

Cardiac catheterization and angiocardiography have shown that the high pressure in the right auricle and superior vena cava is due to the obstruction of the circulation beyond the right ventricle. The size of the right auricle is not reduced, nor is there any obstruction to the superior vena cava, which is increased in size. A good deal of experimental work in dogs has shown that constriction of the superior vena cava above the azygos vein produced no ill effects, but that constriction below this site was followed by pleural effusion. Constriction of the inferior vena cava caused ascites.

Holman and Willett (1949) emphasize the importance of an adequate exposure of the entire heart and great vessels. They report (Holman and Willett 1955) the results of treatment of 26 patients and conclude that pericardiectomy must be radical to be effective and, in order to liberate all critical areas, the decortication must extend beyond the left border, right border, and lower border, with release of both the inferior and superior venae cavae and the base of the heart. They state that limited or incomplete removal of the pericardial scar leads to failure or long delay in the resolution of the ascites, due probably to the persistence of the scar overlying the right ventricle or auricle, superior or inferior vena cava, or even the pulmonary artery at the base of the heart. They state, furthermore, that the pericardium over the left ventricle is never so thickened or so oppressive as the pericardium over the right heart and the two caval vessels.

The figures for post-operative venous pressure published by these authors indicate a persistence of venous hypertension in the majority of their cases.

Harvey *et al.* (1953), in a paper on the mechanical and myocardial factors in chronic constrictive pericarditis, point out that the disturbances could not all be solely ascribed to mechanical restriction of the heart, also emphasize the importance of myocardial function and hypervolaemia.

One would not expect rapid resolution of the hepatic and pulmonary hypertension and of the arterio-sclerotic changes after a wide decortication. The cases published in this present series have shown considerable clinical improvement in spite of a prolonged period of post-operative venous hypertension. One does not know how long it takes for these changes to revert to normal.

Experiments have been carried out to produce pericardial constriction without causing myocardial damage. Isaacs *et al.* (1952), in concluding that constriction of the right auricle is rarely of primary importance in constrictive pericarditis, contend that total excision of the pericardial scar is to be desired and liberation of both ventricles is essential.

Auricular decortication was not performed in the series of cases now under review owing to the attendant dangers and doubtful benefits of this procedure.

Time for Operation

Conflicting views are held concerning the correct time for operation. Of course, clear-cut signs of constriction constitute a clear indication for operation. The patients in many instances will require treatment for congestive cardiac failure before pericardiectomy is considered. This can always be given, since pericardiectomy is never an emergency procedure. Tamponade is the only reason for urgent cardiac decompression. It is not difficult to interpret the signs of pericarditis with effusion causing marked tamponade and requiring urgent pericardial aspiration.

It must be remembered that prolonged raised venous pressure may lead to cirrhotic changes of the liver. Treatment should be provided before these changes become irreversible. Holman and Willett (1951) suggest treatment of active tuberculous pericarditis by pericardiectomy and in support of this view they quote a case in which radical pericardiectomy was performed in the presence of effusion, persistent high fever and an active tuberculous process as disclosed by positive culture. It is my contention that such a case can still revert to normal and would probably have profited by conservative treatment, though once constriction changes show themselves, pericardiectomy is imperative. Repeated removals of effusion may be necessary in

cases of pericarditis causing symptoms of tamponade. Lack of improvement from the very onset of the pericardial decompressions is an indication that the case should be considered as one requiring surgical treatment. In stressing my opposition to routine pericardiectomy of all cases of tuberculous pericarditis and effusion, I would like to place equal emphasis on the harmful results ensuing from delay in pericardiectomy where surgical indications do exist.

Pre-Operative Preparation

Patients should be prepared for operation by means of a good balanced diet, a reduction in salt intake, rest in bed under supervision, and the administration of diuretics and digitalis. Antituberculotic measures should always be instituted even without conclusive evidence of a tuberculous etiology. It is most important to aspirate any marked pleural effusion or ascites immediately before the operation.

The pre-operative preparation and antituberculotic treatment usually lasts for about 6 weeks. This treatment will effect improvement of the patient's condition, but it will not remove the signs of constriction. The raised jugular venous pressure and enlarged liver will not return to normal without operation.

The Operation

There are several different approaches to the pericardium:

1. *The Left Parasternal Incision.* This incision involves the resection of segments of the 3rd, 4th, 5th and possibly the 6th rib near their costo-chondrial attachments. It allows of access to the left pleural cavity, or to the left side of the heart without opening into the pleural cavity if the parietal pleura can be displaced laterally. It is an incision that has little to commend it, because after its closure a large area of chest wall over the heart will be devoid of support from the bony thoracic cage.

2. *Median Sternotomy.* The incision over the anterior chest wall allows the division of the sternum from the level of the 2nd interspace to the xiphisternum. The various types of sternum-splitting chisels that have been devised are unnecessary. Once the sternum has been exposed, a Gigli saw can be passed from one side of the sternum to the other after the pleural layers have been separated from behind with a Doyen's raspatory. Similarly the structures posterior to the sternum can be separated from the sternum itself, from the xiphisternum upwards, by means of a long curved rugine. The Gigli saw is again employed to split the sternum vertically, the whole manoeuvre only taking a few minutes. This exposure, which separates the two halves of the chest wall, allows of direct access to the entire anterior aspect of the pericardium. The incision is easily and firmly repaired with wire sutures.

3. *Bilateral Incision.* This incision was suggested by Johnson and Kirby (1951). It is a transverse incision which opens both pleural cavities, the sternum being divided with rib shears at its lower end. The upper and lower borders are separated by a rib spreader and wide access to the entire anterior aspect of the heart is obtained.

4. *Left Transpleural Intercostal Incision.* This incision provides an approach to the heart through the left pleural cavity. If the ribs are widely spread good access to the ventricular surfaces of the heart is secured. The advantage of this incision is that it is readily repaired, leaving no weakness of the chest wall. Experience of all these incisions has made me select this left transpleural intercostal incision as the method of choice for decortication of the ventricular surfaces. The 5th or even the 6th intercostal space allows full access to the pericardium.

Steps in the Decortication

1. *Mobilization of the Lung.* The lung may be adherent to the pericardium over a wide area and should be stripped free by blunt dissection with a gauze swab on a holder. If this is not possible, division can be attained with scissors or knife in order to display the mediastinal aspect of the pericardium.

2. *Separation from the Sternum.* The pericardium, which may be adherent to the back of the sternum along its entire length, can be separated by the use of gauze-stripping swabs. This exposes the entire anterior and left mediastinal aspect of the pericardium. The pericardium of the patient who has not had adequate pre-operative treatment may show oedema, usually just posterior to the sternum.

3. *Incision of Pericardium.* The surgeon may be cognizant of the actual thickness of the pericardium, as this can be estimated pre-operatively. X-ray views of the chest after an opaque cardiac catheter has been inserted with its tip lying in the right ventricle, will supply the necessary information. The injection of an opaque medium will outline the ventricular chamber, the heart shadows will be outlined by straight X-ray and the space between these two shadows will obviously be occupied by the ventricular wall *plus* pericardium. It is not imperative to obtain this information.

A vertical incision, 1 inch in length, should be made over the left border of the pericardium; this corresponds to the site of the left ventricle. When the incision is about 1/8 inch in depth, it should be inspected, and if it is deep enough, i.e. down to heart muscle, the bluish-purple colour of the muscle will be visible. Appearances resembling fibrous or even cartilaginous tissue are an indication that the incision can be extended deeper into the pericardium.

Eventually the correct depth is reached, or it is believed to have been reached, and at this stage the surgeon should try to find a cleavage plane by undermining the incision. A flap is then raised on both sides of the incision. Cleavage planes are found surprisingly often and are not necessarily the correct ones, as the pericardium constricts by the deposition of layers of fibrous tissue.

The flaps of pericardium should then be removed, converting the incision from the slit into an oval area. If the incision has been made down to the correct layer, i.e. down to epicardium or myocardium, the heart muscle will then bulge into this oval-shaped area, as though the captive heart is attempting to extricate itself. The presence of the heart muscle is an indication that the correct layer has been reached, and this may be confirmed by the presence of branches of the coronary vessels. The pericardium should then be removed over both

ventricles. Flaps of pericardium are cut away after they have been undermined with the blades of right-angled clamps, such as those of the O'Shaughnessy type.

Numerous scattered granular tubercles may be visible on the underlying heart muscle.

The whole of the left ventricle should be stripped before stripping is commenced on the right side and extended around the apex of the heart, giving this important part freedom of movement.

During the decortication of the posterior part of the heart, electrocardiographic changes may indicate cardiac ischaemic changes, due to traction on the coronary vessels to this area, and it may be deemed wise to abandon the stripping at this site. The value of continuous ECG tracings during the operation has been fully proved in several cases.

The decortication should be carried as far as the atrioventricular grooves and the auricles and the great vessels should be left undisturbed.

Small collections of pus may be found shut off in the obliterated pericardial sac. Frequently, the entire pericardium can be removed from the ventricles, leaving the epicardium still attached to the heart muscle. If wrinkling of this layer of epicardium is seen during cardiac contractions, it should be removed if possible. Removal of the pericardium only, may prove sufficient in cases of thickened pericardium with effusion. If, however, there is any constricting effect from the thickened epicardium, it too must be removed.

Plaques of calcium may have to be cut off with shears. These plaques may extend into the myocardium and it is not wise to attempt their removal if there are attendant hazards.

There is usually very little blood-loss. The superficial branches of the coronary vessels are clearly visible and their exposure during the early stage of the pericardial removal provides evidence that the right layer has been reached. The likelihood of penetrating the ventricular muscle is remote. This fact is not applicable to the auricular muscle if decortication is attempted at this site. The entire pericardium is affected in the pericarditis process and theoretically it would be desirable to remove all the diseased tissue. This, however, is not necessary because the auricles are usually minimally affected and the stripping of their surfaces is a dangerous procedure.

THE PRESENT SERIES OF CASES

The cases reported in this paper have been treated by decortication at the Groote Schuur Hospital, Cape Town, during the period 1952-55. They were only referred for treatment when the physicians considered the time appropriate for surgery.

The following chart summarizes the distribution by age, race and sex.

Age	*European*		*Coloured*		*African*		*Total*
	M	*F*	*M*	*F*	*M*	*F*	
0- 9 ..			1				1
10-19 ..			2		2		4
20-29 ..	1		2		2		5
30-39 ..			2	1	4		7
40-49 ..			3		7		10
50-59 ..			1		2		3
							30

Dramatic improvement has been the exception in the cases reported in this paper, but very great clinical improvement, which has been maintained and continued over many months, has been the usual result. Sellors (1946) has shown that in such cases the venous pressure often persists at a high level without causing the patient much inconvenience. The following information is a short analysis of the cases which have undergone operation:

No. of patients	30
No. of operations	32
Results:	
Excellent	20
Good	4
Condition unchanged	2
Died at operation	0
Late death	4

It will be noted that 2 patients required a repeat operation. One of these was so ill at the time of the first operation that only an inadequate amount of pericardium could be removed. Temporary improvement was followed by a recurrence of all the symptoms. The other patient rapidly developed constrictive signs after operation. In both cases, the heart was encased in a new 'scar'. It is very likely that, in both cases, the original pericardiectomy had not been done deeply enough, and that the constricting epicardium had been left around the heart. In both cases post-operative aspiration of ascites was performed after the first operation, but was not necessary after the second operation.

A mixture of penicillin and streptomycin powder was inserted into the pleural cavity on the left side in all cases and a self-retaining catheter was placed in the pleural cavity in order to drain away any excess blood and secretion. It was noteworthy that all cases lost between 1,200 and 2,000 c.c. of blood-stained fluid. The surgical rule of drainage of the chest after a thoracotomy is particularly important after a pericardiectomy. Removal of effusions by aspirations is a very inadequate procedure for the patient post-operatively. In my opinion the rapid re-expansion of the lung plays an important part in providing the heart with a 'new pericardium'.

Several surgeons have reported unfortunate mental sequelae in patients following pericardiectomy. This may well be due to cerebral anoxia from an over-dilated heart which has lost the restraining action of the pericardium. All patients must be given excess oxygen through nasal catheters and masks for 3-4 days, particularly if the jugular venous pressure appears to be on the increase.

Post-Operative Drugs

Therapy with antituberculotic drugs should be continued. Streptomycin should be given daily and INH should be administered by mouth for one month at least. The streptomycin should then be given bi-weekly. Adequate doses of digitalis are given and two injections of Mersalyl or other diuretic per week. This procedure was followed in the present series.

Results

With these above measures the progress has usually been steadily maintained in the 30 cases. Four deaths took place, none at the time of operation. One occurred on the 21st post-operative day from pulmonary embolism, and it is interesting to note in this case that difficulty was experienced during the induction of anaesthesia, the patient going into a state of pulmonary oedema before the commencement of the operation; he showed signs of extreme cerebral anoxia after recovery from the operation and severe mental sequelae preceded his death. Another patient, who died from tuberculous septicaemia 4 weeks after operation, had undergone a sternum-splitting approach, and the incision broke down on the 3rd day, requiring repair. Another died 4 days after operation from pulmonary oedema; there was also extensive chronic lung fibrosis. A Cape Coloured patient, who suffered from epilepsy, developed signs of mental excitement post-operatively, which necessitated his transfer to a mental hospital. The physiological changes following operation indicated that the pericardiectomy had been successful, but 3 months later he died from a toxic psychosis.

Post-operative aspiration of ascites was performed in the two patients who developed constrictive signs after the first operation, but was not necessary after the second operation.

All the cases were transferred to the care of the medical staff when their recovery from the operation was complete. Reports from the physicians in charge of the cases are in the process of compilation.

This survey of the cases so far treated in this Surgical Unit justifies repetition of the statement made by Schmieden in 1926 and 1937:

1. Liberate the left ventricle first.
2. Next free the right ventricle.
3. The auricular walls are thin and should not be freed.
4. Special attention must be paid to the apex of the heart, with sacrifice of the left phrenic nerve if it cannot be freed.

The majority of the cases reported in this paper were treated at the Groote Schuur Hospital. The pre-operative preparation was managed by Dr. V. Schrire who provided electro-cardiographic assistance during the operations.

REFERENCES

Bailey, C. P. (1955): *Surgery of the Heart.* Philadelphia: Lea & Febiger.
Blalock, A. and Burwell, C. S. (1941): Surg. Gynec. Obstet., **73,** 433.
Decker, P. (1947): Presse Méd., **55,** 59.
Evans, W. and Jackson, F. (1952): Brit. Heart J., **14,** 53.
Harvey, R. M., Ferrer, M. I., Cathcart, R. T., Richards, D. W. and Cournand, A. (1953): Circulation, **8,** 695.
Holman, E. and Willett, F. (1949): Surg. Gynec. Obstet., **89,** 129.
Idem. (1951): J. Amer. Med. Assoc., **146,** 1.
Idem. (1955): *Ibid.*, **157,** 789.
Johnson, J. and Kirby, C. K. (1951): Ann. Surg., **133,** 540.
Klinkenbergh, M. C. (1951): J. Thorac. Surg., **19,** 684.
Moschowitz, E. (1953): J. Amer. Med. Assoc., **153,** 194.
Schmieden, V. and Fisher, H. (1926): Ergebn. Chir. Orthop., **19,** 98.
Schmieden, V. and Westermann, H. H. (1937): Surgery, **2,** 350.
Sellors, T. H. (1946): Brit. J. Surg., **33,** 215.
Shore, L. R. (1929): J. Anat. (Lond.), **63,** 291.

IN MEMORIAM

THOMAS BENJAMIN DAVIE

Professor M. van den Ende, of Cape Town, writes: The death in London on 13 December 1955 of Dr. T. B. Davie, who since 1948 was Principal and Vice-Chancellor of the University of Cape Town, has come as a blow to the academic world in South Africa.

Dr. T. B. Davie

Thomas Benjamin Davie was born on 23 November 1895 in Prieska, Cape Province. Having completed his B.A. degree with honours at the Victoria College in 1914 he continued his studies at Stellenbosch and obtained the First Class Professional Teachers' Certificate with distinction in 1916. He then taught in schools in the Orange Free State and Transvaal until 1924 when he proceeded overseas to study medicine. The M.B., Ch.B. degree of the University of Liverpool was awarded to him with first class honours in 1928 and the M.D. in 1931. In the same year he obtained the Membership of the Royal College of Physicians, and in 1940 the Fellowship of the College was awarded to him.

His achievements as a scholar were of the highest order, and it is not surprising that the University from which he graduated soon appointed him to a lectureship. Within 7 years of graduating in Medicine he was appointed to his first chair—that of Pathology and Bacteriology at Bristol, and 3 years later, in 1938, he returned to Liverpool as the George Holt Professor of Pathology. He relinquished that chair in 1945 when he became full-time Dean of the Faculty of Medicine at Liverpool, but maintained contact with his subject as Professor of Applied Pathology.

Dr. Davie served as a 2nd Lieutenant in the R.A.F. during the first world war. He was wounded while serving with the Transvaal Scottish Regiment in Johannesburg during the strike in 1922. In the last war his ability as pathologist and administrator was used to the full—he was adviser in Pathology to the Ministry of Health, consultant in Pathology to the Ministry of Supply and the Admiralty and a Regional Blood Transfusion Officer.

As a pathologist he made significant contributions. His teaching laid a sound foundation for the many medical students with whose training he was concerned and an impressive list of publications is evidence of his contribution to knowledge in his subject.

Dr. Davie's appointment in 1948 to the post of Principal and Vice-Chancellor of the University of Cape Town meant a loss to the University of Liverpool with which he had been associated for so many years, but it was to prove an enormous gain for the South African universities and more particularly that in Cape Town. He soon became known as the courageous and outspoken protagonist of all that a university should stand for. His published works since he became Principal are few, but his pronouncements made in his official and private capacities soon made him known throughout this country and overseas. He fearlessly upheld the interests of the universities and the principles of academic freedom; and foremost amongst these he placed the right of the university

to decide what to teach, who should teach, and who should be taught.

Although he retained his love for Medicine and therefore his interest in the medical school, he never forgot that the proper functioning of a university required a careful balance between the Arts and Sciences on the one hand and Professional Studies on the other. Only such a properly balanced university could effectively serve the state and the world in the fullest sense of the term. With his own experience as a student in Arts and Science, as an educationist, in the profession of Medicine, and as an administrator, he was in a strong position to guide his University along the correct path.

Dr. Davie's assistance was sought by many institutions and organizations, both public and private. As a member of the South African Council for Scientific and Industrial Research and Chairman of its Medical and Dental Research Committee, he assisted with the advancement of scientific research in South Africa, medical research in particular benefiting by his membership of these bodies. As member of the National Council for Social Research he threw his weight also into the development of research in other fields. Since the recent appointment of an advisory committee to the Minister of Education, Arts and Science, Dr. Davie has served on it as the representative of the Committee of Principals. No worthier or more energetic champion of the universities could have been found for this important position. In all these councils, as in the many University committees on which he served, it was in particular his ability to grasp the gist of a problem and to clarify it for others that made him such a valuable member and which made it possible for him to achieve so much as an administrator.

Dr. Davie's service to higher education has gained recognition in South Africa and overseas. He was a Fellow of the Royal Society of South Africa. The University of Cambridge conferred on him the honorary degree of LL.D., and an honorary doctorate was to have been conferred on him by the University of Natal. During his last visit to Britain, where he represented his University at the installation of the Queen Mother as Chancellor of the University of London, his old University and the University of Oxford both made known their decision to award honorary doctorates to him. Unfortunately his illness prevented him from receiving these honours but he had the satisfaction of knowing that his work had been considered worthy of such further recognition.

His capacity for work has made Dr. Davie's name almost proverbial. In spite of prolonged serious illness which would have incapacitated lesser men he undertook the full burden of his official duties. In addition he was called upon to play a leading part in many public functions, which encroached upon his limited leisure. In spite of his many duties he remained one of the most approachable men at the University. He was never too busy to see a member of his staff or of the student body, to listen to their problems and to offer his encouragement and guidance. He found time also for a wide range of cultural pursuits and hobbies, including philately, and to offer at his official residence a typical South African hospitality, not only to his own friends and colleagues, but to the many official visitors to his University. Many an industrialist, educationalist and scholar will have vivid memories of the hospitality so liberally dispensed at Glenara. In all his activities Dr. Davie received the devoted assistance of his wife, Mrs. Vera Davie (née Roper), who through her own work and her unselfish devotion to her husband has added to the stature of the Vice-Chancellorship of the University of Cape Town.

Dr. Davie was born a South African and, although for 20 years he lived in Britain, he remained a South African at heart. He was keenly interested in his country, its peoples and their problems, its flora and its fauna. With his death South Africa has lost an outstanding citizen and the University of Cape Town a servant and friend which it will find hard to replace.

PUBLICATIONS BY DR. T. B. DAVIE

Books

A New Dictionary for Nurses. With Lois Oakes, S.R.N. Messrs. E. & S. Livingstone, Edinburgh. Pp. 471, illustrated. 1st edition 1932, 9th edition 1946.

A Pocket Medical Dictionary (based on above). With Lois Oakes, S.R.N. Messrs. E. & S. Livingstone, Edinburgh. 1st edition 1933, 7th edition 1946.

Pathology. An Introduction to Medicine and Surgery. With J. H. Dible, M.B., F.R.C.P. J. & A. Churchill, London. Pp. 946, with 395 illustrations. 1st edition 1939, 2nd edition 1945.

Papers

A Case of Suppurative Pylephlebitis Secondary to Carcinoma at the Duodenal Biliary Papilla (with J. Crosbie Ross, 1931): Brit. J. Surg., **19,** 74, 336.

The Pathology of Intercranial Neoplasms (1932): Lpool Med. Chir. J., **40,** 1.

Medullo-epitheliomas of the Brain and Retina (1932): J. Path. Bact., **35,** 359.

Lymphatic Leukaemia with Cutaneous Manifestations (with R. M. B. McKenna, (1932): Lancet, **1,** 1196.

Post-mortem Pathology of Pulmonary Tuberculosis (1933): Lpool Med. Chir. J., **41,** 63.

Hepato-lienography by the Aid of Thorotrast—Its Uses and Dangers (with P. H. Whitaker and F. Murgatroyd, 1933): Quart. J. Med., **2,** 5, 49.

Bilateral Obliteration of Radial and Carotid Pulses in Aortic Aneurysm (with Henry Cohen, 1933): Lancet, **1,** 852.

Renal-adrenal Adherence (1935): Brit. J. Surg., **22,** 87, 428.

Adenolymphoma of the Salivary Glands (with R. Carmichael and M. J. Stewart, 1935): J. Path. Bact., **40,** 601.

Tuberculous Verrucose Endocarditis (1936): *Ibid.*, **43,** 313.

Renal Rickets (with N. L. Price, 1937): Brit. J. Surg., **24,** 95, 548.

The Supervention of Osteogenic Sarcoma in Paget's Disease (with W. E. Cooke, 1937): *Ibid.*, **25,** 98, 299.

Massive Fat-replacement of the Pancreas (1938): J. Path. Bact., **46,** 473.

Symmetrical Peripheral Gangrene in Cardiac Failure (with C. Bruce Perry, 1939): Brit. Med. J., **1,** 15.

The Preparation and Use of Dried Plasma for Transfusion (with F. R. Edwards and J. Kay, 1940): *Ibid.*, **1,** 377.

Preserved Blood—An Analysis of Its Use. Report from the Merseyside War Blood Bank (with F. R. Edwards, 1940): *Ibid.*, **2,** 73.

Suprarenal Haemorrhage in Meningococcal Septicaemia (with W. H. Grace and C. V. Harrison, 1940): Lancet, **2,** 102.

Preparation and Use of Concentrated Red Cell Suspensions in Treatment of Anaemia (with G. E. O. Williams, 1941): *Ibid.*, **1,** 377.

Septicaemia and Allied Conditions (1941): Practitioner, **47,** 545.

The Pathology of T.N.T. Poisoning (1942): Proc. Roy. Soc. Med., **8,** 558.

Septicaemia, Pyaemia and Toxaemia (1942): Nursing Mirror, **76,** 53.

Intra-epidermal Carcinoma (Bowen's Disease) of the Vulva (with T. N. A. Jeffcoate and C. V. Harrison, 1944): J. Obstet. Gynaec. Brit. Emp., **51,** 377.

Pathological Basis of Sudden Death (1945): Lpool Med. Chir. J., **49,** 26.

Development of Post-graduate Medical Studies in South Africa (1949): S. Afr. Med. J., **23,** 675.

The University Library (1952): S. Afr. Libraries, **20,** 25.

Education and Race Relations in South Africa (1955): Hoernle Lecture, S. Afr. Inst. Race Rel.

DR. T. B. DAVIE. IN MEMORIAM SERVICE AT CAPE TOWN

An *In Memoriam* service for the late Dr. T. B. Davie, Principal of the University of Cape Town, who died in England on 13 December 1955, was held in the Jameson Memorial Hall of the University on 22 December. The service, which was conducted by Rev. P. H. S. Sitters (University Chaplain), Rev. Dr. A. J. van der Merwe (Moderator Dutch Reformed Church), and Rev.

Prof. I. Abrahams (Chief Rabbi), was most impressive; the congregation exceeded a thousand.

The Chancellor and the members of the University Council and Senate, and others, moved in solemn procession to their places to the strains of Chopin's Funeral March, played on the organ by Prof. Dr. Erik Chisholm, who also played Handel's Dead March in Saul on their retiral at the end of the meeting. After a prayer, Psalm 23 was read, hymns were sung, and solos from oratorio. The music was under the direction of Dr. Chisholm.

An Address was delivered by the Hon. Mr. Justice A. v. d. S. Centlivres, Chancellor of the University, who referred to Dr. Davie's sterling qualities, his active and magnetic personality, and his enquiring and truth-seeking mind. During his 7 years' principalship he had rendered eminent service to the University. He had become the embodiment of what the University stood for, to such an extent that it was difficult to envisage the University without him.

An outstanding feature of Dr. Davie's character was his breadth of outlook. His interests were not confined to the University, and he devoted himself heart and soul to furthering education in all its forms. He held that the task of education was not merely to teach technical skills but to enable the youth of the nation to make full use of their minds in order to meet the problems of humanity. The individual should not blindly accept ready-made opinions, but should seek out truth for himself. Dr. Davie uttered many warnings against the sectionalizing of education and against the indoctrination of youth.

The foundations of Dr. Davie's own education had been well and truly laid. He took a science degree at Stellenbosch University and studied the humanities there as well. After serving in the first world war he worked in the teaching profession for several years. Though he afterwards took up medicine he always stressed the importance of the humanities. He held that true education was the greatest gift to mankind, and continually advocated that it should therefore be open to all, irrespective of race or creed. Education ought not to cease when a university degree was obtained, but should continue throughout life; that, he held, was the purpose of a degree.

Dr. Davie was one of those great men who realized their duties to the world at large. He was constantly asked to address public gatherings, and had become one of our leading public men. He was an outstanding son of South Africa and one of the greatest educationalists the country had produced. He would be missed by other South African universities as well as Cape Town, and by many friends overseas.

The Chancellor referred to the sufferings Dr. Davie had endured on account of his chronic ailment, in spite of which he had so efficiently carried out his great work. The whole University sympathized with Mrs. Davie who, always at his side, had been a constant source of strength.

> This is the happy warrior. This is he
> That every man in arms should wish to be.

MEDICAL TOUR OF EUROPE—1956

As mentioned in the *Journal* of 7 January, a number of doctors and their wives are planning to visit Europe this year and accordingly tentative arrangements have been made to organize a comprehensive tour starting on 27 May 1956, to take in the B.M.A. Congress at Brighton, which commences on 9 July.

The tour will be designed to provide comfortable means of visiting many European countries and will include sight-seeing excursions and a certain amount of entertainment on the Continent, such as going to operas and seeing cities by night. In addition, arrangements have already been set in train for official entertainments, receptions and programmes by the Ministries of Health and/or Medical Associations of Israel, Italy, France and England.

The tour includes: Travel to Europe and back by air and in Europe by private bus, boat and train; Hotels: First class with private bathrooms; Half pension, i.e. breakfast and one meal—In England bed-and-breakfast only.

The tour will include Israel, Greece, Italy, France, Switzerland, England, Sweden, Norway, Denmark and Holland. Paris will be the last city visited before returning to South Africa by air about 2½ months from the commencement of the tour.

The fare will be not more than £700. Those wishing to continue at extra cost may arrange to go to the USA or anywhere they wish before returning to the Union by air.

Anyone interested in the tour can obtain details from Dr. J. H. Struthers, Room 28, Administrative Building, General Hospital, Pretoria, or from Dr. E. B. Woolff, 34 Jan Smuts Avenue, Forest Town, Johannesburg.

DRUG REGISTERS : MEDISYNE-REGISTERS

At a recent meeting of the Cape Western Branch Council it was reported that medical practitioners are not keeping registers of drugs as required by the Medical, Dental and Pharmacy Act, and the *Journal* has been requested to publish a notice on the subject. Full information was given in an editorial published a year ago (S. Afr. Med. J., **28**, 1053, 11 December 1954), and this article is here re-published in full.

DANGEROUS DRUGS; 4TH, 5TH AND 6TH SCHEDULES

The Medical, Dental and Pharmacy Act, as amended, schedules 3 classes of 'dangerous' drugs, viz. (1) poisons, (2) habit-forming drugs and (3) potentially harmful drugs, which are respectively listed in the 4th, 5th and 6th schedules of the Act. The schedules are not mutually exclusive; certain drugs appear in more than one schedule.

Amongst the obligations imposed on medical practitioners by the amended Act are the following:

Every medical practitioner who dispenses medicine must keep a prescription book in which he is to enter certain particulars concerning any medicine he dispenses which contains either a

By 'n onlangse vergadering van die Raad van die Tak Wes-Kaapland was dit gerapporteer dat mediese praktisyns nie 'n register van medisyne daarop nahou soos verlang word deur die Wet op Geneeshere, Tandartse en Aptekers nie, en die *Tydskrif* is versoek om 'n kennisgewing hieroor te publiseer. Volledige inligting was 'n jaar gelede in 'n hoofartikel verskaf (S. Afr. T. Geneesk., **28**, 1053, 11 Desember 1954) en die artikel word hier in sy geheel herdruk.

GEVAARLIKE MEDISYNE : 4DE, 5DE EN 6DE BYLAES

Die Wet op Geneeshere, Tandartse en Aptekers, soos gewysig, bevat in die 4de, 5de en 6de bylaes respektiewelik, 3 klasse medisyne wat as gevaarlik bestempel is, nl. (1) vergifte, (2) gewoontevormende medisyne en (3) moontlik nadelige medisyne. Die een bylaag sluit nie die ander uit nie en sekere middels verskyn op meer as een bylaag.

Die gewysigde Wet plaas sekere verpligtings op geneeshere, onder ander die volgende:

Elke geneesheer wat medisyne opmaak moet 'n resepteboek aanhou waarin hy sekere besonderhede moet opteken oor enige medisyne wat hy opmaak wat òf 'n vergif,[1] òf 'n gewoontevormende medisyne,[2] òf 'n moontlik nadelige medisyne bevat.[3] Hierdie besonderhede is die naam en hoeveelheid van elke bestand-

poison,[1] a habit-forming drug,[2] or a potentially harmful drug.[3] The particulars that must be entered are the name and quantity of each ingredient of the medicine, the name and address of the person to whom it is supplied, and the date when it is supplied. Although the relative sections leave room for argument, this appears to sum up the intention of the Act as regards the prescription book, and apart from legal requirements it is a wise precaution on the part of the dispensing doctor to keep records in the manner thus laid down.

The medical practitioner who 'uses or compounds or dispenses' any habit-forming drug is required[4] to cause to be entered in a book called the 'register of habit-forming drugs' (to be kept exclusively for this purpose): (*a*) the quantity of such drugs possessed or acquired by him, (*b*) the date of the acquisition, (*c*) the person from whom and the place from which the same was acquired, and (*d*) the quantity disposed of and whether (i) by sale (in which case the date of the sale and the name and address of the purchaser must be entered) or (ii) by dispensing or use in the ordinary course of practice.

There is, however, a proviso[4] that, where the practitioner keeps a prescription book (or a day book) in the manner we have indicated, it is sufficient for him to record in his register the total quantity of each habit-forming drug used for dispensing and the serial number of the relative entries in the day book or prescription book.

The register must be properly kept up to date and must be balanced so as to show the quantity of each habit-forming drug in stock on the last day of every March, June, September and December (the balancing to be completed within 3 days of those dates).[5] The register must be kept and retained for 3 years and is open to inspection by persons authorised in writing as prescribed by the Act.[6]

No habit-forming drug acquired by a medical practitioner shall be used by him except for strictly medicinal purposes.[7] It may not be used for the satisfaction or relief of a habit or craving except in certain approved institutions. It is also an offence for any person to keep in his possession or control any habit-forming drug[8] or any poison[9] without using all reasonable care in its custody or use. Every medical practitioner also 'shall label with the words "Poison" and "Vergif" every vessel, box, bottle, wrapper or package in his possession which contains any article which is a poison'.[10] This requirement, however, does not apply to any medicine dispensed by a medical practitioner if it is labelled with his name and address and the relative particulars are entered in his prescription book[1] (see above).

There is a similar proviso conditionally exempting medicines dispensed by a medical practitioner from the requirement that medicines for external use containing a poison must be in a container distinguishable by touch (i.e. a 'poison bottle') and labelled 'For External Use Only—Alleen vir Uitwendige Gebruik'.[1] Apparently the Act contains no such exemption in respect of potentially harmful drugs;[11] the omission of the exemption would appear to be an oversight.

Finally, amongst the obligations imposed on medical practitioners who dispense, is one introduced by regulations made by the Minister under section 42 of the Food, Drugs and Disinfectants Act, 1929. These regulations prescribe that every package containing any food or drug must be labelled with the name and business address of the . . . person by whom or on whose behalf it was enclosed in the package. 'Drug' is defined as any substance or mixture of substances used as a medicine, whether internally or externally, and it is therefore held that a medical practitioner who dispenses his own medicine must label all the containers with his name and address, irrespective of whether or no the medicine contains poisons or other scheduled drugs.

Passing to certain legal requirements which are the concern of all practising doctors, and not only those who dispense, we end with the following:

Every prescription ordering any habit-forming drug must state the name and quantity of such drug that may be sold or supplied, the name and address of the person to be supplied and of the practitioner signing (as well as the qualification of the latter), and the date of issue of the prescription; and not more than one issue of the drug may be made on the prescription.[12]

The same provisions apply to prescriptions ordering any potentially harmful drug (though not poisons as such), with the difference deel, die naam en adres van die persoon aan wie dit verskaf word en die dag van lewering. Alhoewel daar oor die vertolking van die betrokke artikels meningsverskil kan ontstaan, is die doel, wat die Wet i.v.m. die resepteboek beoog, in dié artikels opgesom en elke verstandige geneesheer sal, afgesien van die vereistes van die Wet, so 'n resepteboek aanhou.

Elke geneesheer wat gewoontevormende medisyne ‚gebruik, opmaak of resepteer' moet in 'n boek genoem die ‚register van gewoontevormende medisyne' (wat uitsluitlik vir hierdie doel gehou word) die volgende besonderhede laat inskrywe:[4] (*a*) die hoeveelheid van sulke medisyne wat hy besit of verkry het, (*b*) die datum waarop dit verkry is, (*c*) van wie en van waar dit verkry is, en (*d*) die hoeveelheid wat van die hand gesit is en of (i) dit verkoop is (in welke geval die dag van die verkoop en die naam en adres van die koper opgeteken moet word) of (ii) dit in die gewone loop van die praktyk opgemaak of gebruik is.

Die Wet bepaal egter[4] dat wanneer 'n geneesheer hierdie besonderhede alreeds in 'n resepte- of dagboek aangeteken het, dit voldoende is as hy in sy ‚register van gewoontevormende medisyne' melding maak van die hoeveelheid van elke gewoontevormende medisyne wat by die opmaak van resepte gebruik is, en van die volgnommer van die betrokke aantekenings in die resepte- of dagboek.

Die register moet tot op datum en in behoorlike volgorde bygehou word en dit moet 'n balans wys wat duidelik aantoon hoeveel van elke soort gewoontevormende medisyne op die laaste dag van Maart, Junie, September en Desember in voorraad is. (Die balans moet binne 3 dae na elkeen van voormelde datums voltooi word).[5] Die register moet vir 'n tydperk van minstens 3 jaar behou en bewaar word en moet ter insage beskikbaar wees aan persone wat skriftelik daartoe ingevolge die Wet gemagtig is.[6] Geen gewoontevormende medisyne wat deur 'n geneesheer verkry is mag andersins deur hom gebruik word nie dan stiptelik as geneesmiddel.[7] Dit mag nie gebruik word nie om 'n gewoonte of drang te bevredig nie behalwe in sekere goedgekeurde inrigtings. Elkeen wat enige gewoontevormende medisyne[8] of enige vergif[9] in sy besit of bewaring hou sonder om elke redelike voorsorg daarvoor te tref, is aan 'n misdryf skuldig. Elke geneesheer moet ook aan ‚alle vaatwerk, elke kissie, bottel, omslag of pakkie in sy besit wat 'n stof bevat wat 'n vergif is 'n etiket heg met die woorde „Vergif" en „Poison" daarop.'[10] Hierdie vereiste geld egter nie vir enige medisyne wat deur 'n geneesheer opgemaak is en wat van 'n etiket met sy naam en adres voorsien is en waarvan die betrokke besonderhede in sy ‚resepteboek' (soos bovermeld) aangeteken is.[1]

Daar is 'n soortgelyke bepaling wat onder sekere omstandighede die geneesheer wat medisyne opmaak vrystel van die vereiste dat medisyne vir uitwendige gebruik, wat 'n vergif bevat, in 'n houer moet wees wat deur betasting onderskei kan word (d.w.s. 'n ‚gifbottel') en wat voorsien moet wees van die opskrif ‚For External Use Only—Alleen Vir Uitwendige Gebruik'.[1] Oënskynlik maak die Wet geen voorsiening vir so 'n vrystelling in verband met moontlik gevaarlike medisyne nie;[11] dit is waarskynlik 'n glips.

Ten slotte is daar onder andere die regulasies kragtens Artikel 42 van die Wet op Voedingsmiddels, Medisyne en Ontsmettingsmiddels van 1929 wat geneeshere wat medisyne opmaak verplig om elke pakkie wat kos of medisyne bevat te voorsien van 'n etiket met die naam en besigheidsadres van die persoon deur wie of ten behoewe van wie dit verpak is. ‚Medisyne' word gedefinieer as enige stof of mengsel stowwe wat, inwendig of uitwendig, as geneesmiddel gebruik word. 'n Geneesheer wat sy eie medisyne opmaak moet derhalwe alle houers voorsien van etikette met sy naam en adres, afgesien daarvan of die medisyne vergifte of ander medisyne wat in die bylaes vermeld is, bevat.

Daar is sekere regsvereistes wat op alle praktiserende geneeshere, of hulle medisyne opmaak of nie, van toepassing is en ons sluit met die volgende af:

Elke voorskrif vir 'n gewoontevormende medisyne moet melding maak van die naam en hoeveelheid van sodanige medisyne wat verkoop of gelewer mag word, die naam en adres van die persoon vir wie die medisyne bedoel is en die naam en adres van die geneesheer wat dit onderteken het (en ook sy kwalifikasie), en die datum waarop daardie resep uitgereik is; nie meer as een uitgif van die medisyne mag kragtens die voorskrif verstrek word nie.[12]

Dieselfde bepalings geld vir voorskrifte wat enige moontlik gevaarlike medisyne (maar nie ‚vergifte' as sulks nie) bestel met die onderskeid dat die voorskrif vir moontlik gevaarlike medisynes

that the prescription containing the potentially harmful drug is to state the number of times and the intervals at which the prescription may be dispensed, and (except for medicine for external use) the amount and frequency of each dose. Nevertheless, in emergencies any potentially harmful drug may be sold or supplied on the verbal instructions of a medical practitioner given direct to the chemist, if the practitioner is personally known to him; in which case the practitioner is obliged within 24 hours to furnish the chemist with a written prescription complying with the requirements of the Act.[12]

REFERENCES TO THE MEDICAL, DENTAL AND PHARMACY ACT NO. 13 OF 1928 (AS AMENDED)

1. Sections 55 and 56.
2. Sub-section 6 of section 65.
3. Sub-section 2 of section 65 *bis*.
4. Sub-section 6 of section 65.
5. Sub-section 7 of section 65.
6. Sub-section 8 of section 65.
7. Sub-section 2 of section 65.
8. Sub-section 2 of section 61.
9. Section 54.
10. Section 49.
11. Sub-section 1 (*b*) of section 65 *bis*.
12. Sub-section 5 of section 65.
13. Sub-sections 1, 5 and 6 of section 65 *bis*.

ook melding moet maak van die aantal male en met welke tussenposes die resep opgemaak mag word, en (behalwe in die geval van medisyne vir uitwendige gebruik) die grootte van en hoe dikwels elke dosis geneem moet word. In 'n noodgeval kan moontlik nadelige medisyne egter aan enigeen verkoop of gelewer word op regstreekse, mondelingse opdrag van 'n geneesheer aan 'n apteker aan wie die geneesheer persoonlik bekend is; in so 'n geval moet die geneesheer binne 24 uur 'n geskrewe voorskrif aan die apteker gee wat in alle opsigte aan die vereistes van die Wet voldoen.[13]

VERWYSINGS NA DIE WET OP GENEESHERE, TANDARTSE EN APTEKERS NR. 13 VAN 1928 (SOOS GEWYSIG)

1. Artikels 55 en 56.
2. Sub-artikel 6 van artikel 65.
3. Sub-artikel 2 van artikel 65 *bis*.
4. Sub-artikel 6 van artikel 65.
5. Sub-artikel 7 van artikel 65.
6. Sub-artikel 8 van artikel 65.
7. Sub-artikel 2 van artikel 65.
8. Sub-artikel 2 van artikel 61.
9. Artikel 54.
10. Artikel 49.
11. Sub-artikel 1 (*b*) van artikel 65 *bis*.
12. Sub-artikel 5 van artikel 65.
13. Sub-artikels 1, 5 en 6 van artikel 65 *bis*.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY—ASSISTANT EDITOR

Applications are invited from medical practitioners for the post of Assistant Editor in the service of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale attaching to the post is £1,250×50—1,750 per annum, plus an annual cost-of-living allowance of £176 for single men and £352 for married men. The commencing salary will be determined according to journalistic experience.

The successful applicant must contribute to the Association's Superannuation Fund. He will also be expected to assume duty as soon as possible after appointment.

Applications must reach the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town, on or before 2 March 1956.

A. H. Tonkin
Secretary

Medical House
Cape Town
23 December 1955

VAKATURE—ASSISTENT-REDAKTEUR

Aansoeke word van geneeshere ingewag vir die betrekking van Assistent-Redakteur in diens van die Mediese Vereniging van Suid-Afrika, by die Hoofkantoor te Kaapstad.

Die salarisskaal aan die pos verbonde is £1,250×50—1,750 per jaar, plus 'n jaarlikse duurtetoeslag van £176 vir 'n ongetroude en £352 vir 'n getroude man. Die aanvangssalaris sal volgens joernalistieke ondervinding bepaal word.

Die suksesvolle kandidaat moet by die Vereniging se pensioenskema aansluit. Hy sal ook verwag word om so spoedig moontlik na aanstelling diens te aanvaar.

Aansoeke moet die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, bereik vóór of op 2 Maart 1956.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
23 Desember 1955

PASSING EVENTS : IN DIE VERBYGAAN

Dr. I. B. Taylor, F.R.C.S., Ophthalmic Surgeon of Johannesburg, has been appointed an active member of the Association for Research in Ophthalmology, Inc.

* * *

Dr. C. W. Coplans, specialist in physical medicine, has changed his rooms to 906 Sam Newman House, 28 Burg Street, Cape Town. Telephones unchanged: 2-8080 and 2-5187.

* * *

Meeting on Cell-Growth Research Biologists from 9 countries met on 12-16 December in Paris to study means of encouraging research at the international or regional levels on normal and abnormal cell-growth.

Problems of cell growth, multiplication and differentiation are at the foundation of all forms of life. While the research programme under consideration is intended to investigate only the fundamental processes of the cell, this research may also shed light on certain abnormalities such as cancer.

The meeting was called by the Council for International Organizations of Medical Sciences upon the request of UNESCO. Specialists from research institutes in France, the German Federal Republic, India, Israel, the Netherlands, Sweden, UK, USA and USSR were invited to attend.

International organizations represented, in addition to Unesco and the CIOMS, included the World Health Organization, the International Union of Biological Sciences and the International Union Against Cancer.

BOOK REVIEWS : BOEKRESENSIES

PHYSIOLOGY

The Physiological Basis of Medical Practice. A Text in Applied Physiology. Sixth Edition. By Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (Lond.), F.R.S., F.R.C.P. (Canada) and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada). Pp. 1357 + xiii, with 601 illustrations. 91s. 6d. London: Baillière, Tindall & Cox Ltd. 1955.

Contents: 1. The Blood and Lymph. 2. The Circulation of the Blood. 3. Respiration. 4. The Excretion of Urine. 5. Digestion. 6. Metabolism and Nutrition. 7. The Ductless Glands or Endocrines. 8. The Nervous System. 9. The Special Senses. The Chemical Senses. References. Index.

'Best and Taylor' does not need much reviewing—it is too well known—nor is it possible adequately to review a vast textbook covering the whole of physiology, and not only pure physiology, but its application to medicine. Actually, it seems to me, a pure physiology book is fairly simple—it presents facts and theories based directly on those facts. On the other hand a book of this type must also mention, and briefly describe, pathological states and then attempt to explain them on the basis of altered physiology. In other words, it must write a textbook of medicine in terms of physiology. In this 'Best and Taylor' does not quite succeed. The physiology is authoritative and most admirably up to date (e.g. 'electrocortin' appears, and there is discussion on Mayer's glucostatic theory of hunger), but its medicine is less so. Thus Cushing's syndrome appears among the diseases of the pituitary, and the pituitary still gets blamed for other states which it cannot produce (e.g. obesity). Several medical conditions which would seem most admirably to be suited to physiological discussion do not appear (e.g. the congenital and acquired specific renal tubular disorders; the small intestinal deprivation, loop and stricture syndromes). I was also disappointed to find no discussion on that increasingly important topic, the altered physiology of ageing, while the mechanisms and applications of heredity are not considered.

I should not think the junior physiology student could find this book easy reading, and of course, the medical applications will be lost on him. It must be more as a work of reference and a textbook for the senior student that it will be valuable. The paper is thin and of moderate quality, while the figures are not always very clear.

P.J.

MEDICAL BELLES LETTRES

Mysterious Waters to Guard (*Essays and Addresses on Anaesthesia*). By Wesley Bourne. Pp. 398 + xvi. 42s. Oxford: Blackwell Scientific Publications. 1955.

Contents: 1. Some Recent Advances, Presidential Address, 1925. 2. An Estimate of the Usefulness of some of the Newer Anaesthetics. 3. Anaesthetics and Liver Function. 4. Analgesia and Anaesthesia in Obstetrics. 5. The Perfecting of Anaesthesia. 6. Anaesthesia for the Republic of Plato. 7. *Fors Clavigera*. 8. Factors Determining Selection and Administration of Anaesthesia. 9. Trends in Inhalation Anaesthesia. 10. Francis Hoeffer McMechan: An Apotheosis. 11. Anaesthesia in Perspective. Presidential Address, 1939. 12. Choice of Anaesthesia in Emergency Surgery. 13. *De Officiis*. 14. Anaesthesia in War Circumstances. 15. Breathing in Anaesthesia. 16. Anaesthesia for the Tubercular Patient. 17. Presidential Address: American Society of Anaesthetists, 1942. 18. Interdependence of Function. 19. Anaesthesia: Some Contemplations, Osler Memorial Lectures. 20. Pure Ether and Impurities: A Review. 21. Gaseous Anaesthetics, Morton Centennial. 22. Impressions of the Centenary Celebrations of Anaesthesia in Great Britain. 23. Wise Indifference of the Wise. 24. Pursuit of Learning. 25. What's New. 26. At the Head of the Table. 27. On Learning Anaesthesia. 28. The Role of Basic Sciences. 29. Measure and Order. 30. Essentials of an Efficient Anaesthesia. 31. Recent Advances, 1950. 32. *Discours d'ouverture du Centre d' Anesthésiologie de Paris* 33. Cultivate your Garden. Epilogue.

In this collection of essays and addresses, covering the period 1925-50 and culled from a much larger personal contribution to the literature of anaesthesia, of physiology and pharmacology, and of medicine, Wesley Bourne, Emeritus Professor of Anaesthesia at McGill University and doyen of Canadian anaesthetists, emphasizes indirectly a loss widely felt in our profession and in the world at large—the loss of that indefinable characteristic implied in Cardinal Newman's conception of an educated man.

While these essays contain ample material to illustrate the author's grasp of the fundamental facts pertaining to anaesthesia and to all the other fields of medicine, many of them facts in the establishing of which he himself played an important part, they contain far more than that. They exhibit overwhelming evidence of the fact that the author is one who is widely read and who 'loves to think'. Practitioner, teacher, guide and philosopher, Bourne has gathered together a diadem of sparkling literary effort—sparkling with his own judiciously turned phrases and with gems borrowed from classical and modern literature.

This book will be most valuable to those who are interested in the development of the speciality of anaesthesia over the ¼ century which it covers, not only for the documentation of that development and for the portrait gallery of personalities in anaesthesia (South Africa is not excluded here) but for the intimate picture it gives of one of those who are most notable in this period. It will be valuable too, to those who wish to learn again the lesson of relativity, for many an early and very dogmatic prognostication has been disproved by time and circumstances. But the volume has a value over and above such mundane considerations.

Those who feel (and they need not be anaesthetists) that the urgent demands of modern medical practice have condemned them to a life deprived of that peace of mind which the leisurely perusal of a well-loved classic will bring; a life deprived of that depth of purpose which a wide, a well rounded and an ever-expanding education provides; a life of a slave thinly disguised by the floridity of 'a highly trained professional man', will find an example in this collection of the life they yearn for, and perhaps the courage to pursue it.

C.S.J.

ATLAS OF MUSCULO-SKELETAL EXPOSURES

An Atlas of Musculo-skeletal Exposures. By H. F. Moseley, M.A., D.M., M.Ch. (Oxon.), F.R.C.S. (England and Canada), F.A.C.S. Pp. 235 + xii, with 439 illustrations. £9 0s. 0d. Philadelphia and Montreal: J. B. Lippincott Company. 1955.

Contents: 1. General Considerations. 2. The Face. 3. The Clavicle and its Articulations. 4. The Shoulder. 5. The Arm. 6. The Elbow. 7. The Forearm. 8. The Wrist. 9. The Hand. 10. The Trunk. 11. The Hip. 12. The Thigh. 13. The Knee. 14. The Leg. 15. The Ankle. 16. The Foot. Index.

This well-produced book by Professor Moseley of McGill University is a valuable addition to the general surgeon's library on trauma. It comprises a series of fine colour-drawings, illustrating musculo-skeletal exposures of the whole body, and opposite each group of illustrations is a page of text briefly describing the procedures.

The common meeting ground of the general surgeon and the orthopaedic surgeon is the field of trauma—the closed and open treatment of fractures, the treatment of major wounds involving skin loss, tendon loss and bone damage, and late repair by grafting. The opening chapter has a long excerpt from Lane's original writings on asepsis, and a reproduction and discussion of Langer's pictures on skin tension lines, both these stressing fundamental principles of traumatic surgery.

There are particularly useful introductions to each system, wherein the towelling and positioning of the patient are shown in clear black-and-white photographs, and the areas of skin preparation are depicted. These important details are almost always omitted in standard works. A further helpful feature is the addition of a bibliography of films available on each subject and their source of supply.

The section illustrating exposures of facial and temporo-mandibular joints are good diagrammatic representations of the anatomy in this region; and the excellent section on exposures of the shoulder reflect one of Professor Moseley's chief interests. The various methods of approach to the hip-joint are instructive, and the section on exposures of the vertebrae at various levels is clear and concise.

It seems unfair to criticize so satisfactory a production. However, it may be mentioned that, in the text, identification of some diagrams by small letters makes it a little difficult to follow, while in some parts anatomical accuracy has been sacrificed to diagrammatic representation. The incision illustrating the opening of a finger from the lateral aspect should preferably leave skin bridges at the creases.

The price is almost prohibitive, as is usually the case with American publications, but this book forms so natural a complement to Henry's book on the subject, that it will appeal strongly to the surgeon interested in this branch of surgery.

A.E.K.

SURGICAL FORUM

Surgical Forum: Proceedings of the Forum Sessions, Fortieth Clinical Congress of the American College of Surgeons, Atlantic City, N.J. November 1954. By various contributors. Pp. 851 + xi, with illustrations. Philadelphia and London: W. B. Saunders Company. 1955.

Contents: 1. Heart and Great Vessels. 2. Blood Vessels and Circulation. 3. Vascular Grafts. 4. Esophagus, Stomach and Intestine. 5. Liver and Pancreas. 6. Nutrition, Body Fluids and Metabolism. 7. Steroids and Cancer. 8. Anesthesiology. 9. Burns. 10. Shock and Wounds; Plastic Anatomic Cast. Indices.

This authology of up-to-the-minute surgical research should be read, at least by every person engaged upon such matters. It raises the eyes to the hills of surgical inspiration. Every professor of surgery should keep it to hand, and no medical library can afford to be without it. It boils over with ideas, and there is probably no problem in modern surgical research which is not in some way related to, or touched upon by, some or other experiment or idea in this collection of 163 articles by 400 contributors.

The editorial committee contains some of the most respected names in contemporary American surgery, and this gives the book a definite authenticity.

Probably no one man could review the content of this book authoritatively, but without being presumptuous one may criticize a lack of arrangement of the material of the book into some sequence which will promote understanding of the procedures described. This is aggravated by the almost total shortage of technical detail of the pre-operative and post-operative care of the experimental animal, and often of the very procedure described. No doubt this is assumed to be common knowledge among skilled research-unit personnel, but the value of the book would be very greatly increased by a chapter of detailed instruction about this side of the problem—a sort of advanced grade of Sherrington's 'Experimental Physiology'.

The quondam isolation of surgery from its sister-sciences of biology, biochemistry and biophysics is disappearing fast, its horizons are extending rapidly, and nowhere could this be better shown than by such a book as this.

R.D.H.B.

CORRESPONDENCE : KORRESPONDENSIE

DR. SICHEL'S NEW YEAR MESSAGE

To the Editor: May I, through the medium of your *Journal*, congratulate Dr. Alan Sichel, Chairman, Federal Council, on his courageous, inspiring, and timely message to members of the Medical Association of South Africa and to assure him that large numbers feel like him, 'greatly perturbed about the way in which we as a profession are tending towards commercialism'.

Unless this tendency is checked, the day is not far distant when members who honour the history and age-old traditions of the profession of medicine will refuse to serve on Federal Council or other committees that cater 'mainly for the financial status of the individual'.

R. Lance Impey

Claremont, Cape
3 January 1956

1. Sichel, A. W. S. (1955): S. Afr. Med. J., **29**, 1246 (31 December).

ILLNESS CAUSED BY MEDICATION BY WITCH-DOCTORS

To the Editor: A recent issue of the *Journal* contained an interesting report[1] concerning a witch-doctor's conviction on a charge of manslaughter. This followed tetanus contamination of his 'curative' scratches.

Of late a spate of cases has appeared at my surgery of acute toxaemia following oral medication by witch-doctors. A sample of the medicine was sent to the Institute for Medical Research, but was returned with a short note asking for the Public Prosecutor's notice before analysis could be done. Unfortunately, no patient is prepared to charge the witch-doctor; so police procedure is apparently not possible in this case.

My purpose in writing to you is to enquire about a possible method of obtaining analytical indentification of the poison concerned, the better to provide an antidote.

Would you advise me directly or through the *Journal* as to what course I may follow?

T. H. Crouch

Mooi River
30 December 1955

[The Secretary for Health, Pretoria, may authorize analysis in cases of public health interest, such as this case.—*Editor.*)

1. Brink, C. J. H., Visser, C. H. C. and Kahn, M. (1955): S. Afr. Med. J., **29**, 1149 (3 December].

STETHOSCOPE VERSUS ECG

To the Editor: I am entirely *en accord* with Dr. N. Finn[1] on his disagreement with Dr. A. Rabinowitz[2] concerning the use of the electrocardiograph in certain cases. In fact one may well ask whether electrocardiography is not being overdone nowadays. All of us who have been in practice for some time have had the mortification of seeing patients, pronounced OK by eminent cardiologists, succumb shortly afterwards to a coronary calamity; just as we have many patients, given only a few months or years to live after ECG readings, still alive and doing pretty well. I personally am of the opinion that with the intelligent use of a stethoscope one can diagnose and prognose heart trouble with a considerable degree of accuracy. I think in reference to electrocardiography—and this applies equally to other modern medical procedures—we might join chorus with Cicero and exclaim *ubinam gentium sumus.*

H. R. Goldberg

30 Silverton Road
Durban
28 December 1955

FUNCTIONAL AND PSYCHIC ELEMENTS IN HEART DISEASE

To the Editor: In reply to the letter of Dr. Nathan Finn[1] on the above subject I have the following comments to make:

Dr. Finn has taken the reference to X-ray and ECG examinations out of its context. What was stated is the following:[2] 'The importance of differentiating functional from organic heart-disease must be readily appreciated. The absence of cardiac enlargement, organic murmurs, cardiac failure, agina pectoris, abnormal cardiac silhouette on X-ray, or ECG changes, would indicate that organic heart-disease is not responsible for the patient's symptoms or incapacity'.

I do not think that anyone would disagree with this reference to X-ray and ECG examinations where the object is to exclude organic heart disease. Furthermore, few doctors will agree with Dr. Finn's contention that an ECG examination frequently causes functional heart-disease. On the contrary, an ECG examination more frequently convinces the patient that the doctor's opinion has been confirmed by this procedure and that organic heart-disease is, in fact, not present.

Regarding Dr. Finn's reference to disease of the cervical or thoracic spine as a cause of left chest pain, this is of course as well known as are a host of other conditions which may produce left chest pain.

In conclusion, may I thank Dr. Finn for the interest he has shown in the article, and for his kind preliminary remarks?

A. Rabinowitz

908 Medical Centre
Jeppe Street
Johannesburg
3 January 1956

1. Finn, N. (1955): S. Afr. Med. J., **29**, 1244 (24 December).
2. Rabinowitz, A. (1955): *Ibid.*, **29**, 1129 (3 December).